

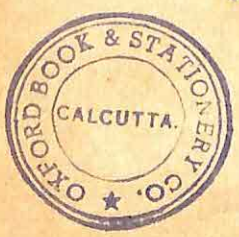
TREATMENT

OF MENTAL DISORDER

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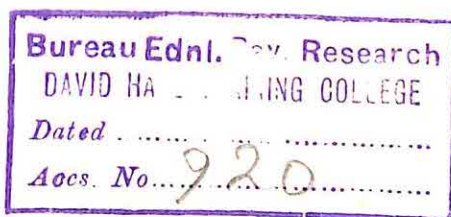


TREATMENT

OF MENTAL DISORDER

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ILLUSTRATED

W. B. SAUNDERS COMPANY

PHILADELPHIA AND LONDON

1953

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This book is written for students of medicine and physicians. It will be of special interest for young neuropsychiatrists about to enter their period of training. In these times of increased awareness of the role of the mind and the emotions in disease, the general physician too may well feel an obligation to widen his knowledge of psychiatric theories and techniques. The emphasis in the following pages on practical means of therapy, controlled by tests for indications and by statistical evaluation of results, may be said to bring psychiatric treatment within a framework of thought similar to the therapeutic approaches of general clinical medicine.

The present book is the product of twenty-three years of practice, teaching and research in the field of nervous and mental diseases. When I was a medical student, what little was known and practicable about the treatment of most mental illnesses could be adequately expressed in the small paragraphs tucked away at the ends of lengthy chapters on diagnosis, phenomenology and interpretation in the standard textbooks of psychiatry. In the past twenty years, however, this picture has changed decisively. Highly effective methods have been developed both in the field of physical treatment, which gave therapeutic optimism in the field of the major mental illnesses its great new impetus, as well as in the field of psychotherapy, based on deepening of psychodynamic understanding. There has been an understandable yet deplorable trend for these two major methods to be developed and written about in isolation from each other. Thus most books on therapy available to the student on this problem today deal with the subject from a one-sided point of view and with utilization of only one approach.

Contest in the struggle for truth, of course, plays a stimulating part in all scientific development. The time has not yet come, however, when this particular contest can be decided on purely theoretical grounds. The cause of most mental diseases is yet unknown, although certain fragments of the interlocking chains of causality have become exposed beneath frequently puzzling and deceptive surfaces. We are not yet in a position to offer a treatment program based on clear-cut recognition of a single cause, applicable to the majority of mental disorders. I have therefore regarded it as an essential preliminary to the writing of this book to collect, tabulate and statistically analyze all the material on treatment results in unselected series of cases that were available to me from the literature. The tabulations included in this book comprise more than 38,000 cases of mental disorder, including 29,000 cases of schizophrenia, the most challenging field of psychiatric therapy today. The analysis of these data reveals sufficiently consistent and reproducible effects, both of physical and of psychologic techniques, to justify surveying the entire field of existing knowledge of the treatment of mental illness in one comprehensive volume.

While for purposes of research the psychiatrist is justified at times in using one approach only, for practical purposes he should bring as many proven effective means as possible to bear against the illness in order to give his patient the maximum therapeutic help that he needs, as promptly as possible. I have attempted, therefore, to integrate the physical and the psychotherapeutic approaches to the treatment of mental disorder, and to show how fruitful such integration may be under actual therapeutic conditions.

The chief purpose of this book is to demonstrate as specifically as possible how psychic and physical aids to psychiatric treatment must be used as one instrument of therapy. Precise prescriptions for the various practical proven techniques are presented and illustrated in detail. Means to their effective mutual integration are presented, and emphasis is given to complications in therapy and their prevention and management.

It is hoped that this book will not only provide a guide for the practical utilization of the therapeutic tools available today to physicians interested in the therapy of mental disorders, but also a sextant, as it were, for determining our present position in the quest for effective therapy, that may aid others in charting the future course in the advance of our science and art.

LEO ALEXANDER

Boston, Mass.
January, 1953

ACKNOWLEDGMENTS

Like every physician since Hippocrates, who incorporated this acknowledgment in his oath, I owe a great deal to my teachers. From Otto Marburg I learned about the intricate structure of the brain and the subtle marks of its pathologic changes. By Paul Schilder, one of my most inspiring instructors in medical school, I was first acquainted with genetic and dynamic psychology and psychiatry. Cécile and Oskar Vogt impressed me with the importance of genetic and constitutional factors. From Maximilian Rose and Eduard Beck, whom I met in the Vogts' Institute, I learned about the rich variability of the cortical cytoarchitecture, and they also instilled me with interest in the older psychotherapeutic techniques, especially Du Bois and Déjérine's persuasive technique and hypnosis. By Karl Kleist I was introduced into the traditions of Wernicke's school, and from him I learned that mental as well as emotional aberrations have physical correlates. From Richard S. Lyman, with whom I was associated in China at Peiping Union Medical College and in North Carolina at Duke University, I learned to appreciate the importance of social factors in eliciting, evoking, enhancing, or relieving mental disorder. I learned more about the influence of social situational stress and motivation on active Army duty overseas, and later at the War Crimes Trials in Nurnberg. From R. G. Hoskins and his group in Worcester I learned about the significance of the bodily state and its biochemical characteristics in reflecting and influencing the mental state. I owe a great deal to the therapeutic genius of Abraham Myerson, with whom I was closely associated from 1935 until his untimely death in 1948. From him I learned about the efficacy of the multifactorial approach, the treatment of the total personality, mental and physical. From Tracy J. Putnam I acquired respect for the persistence, courage, ingenuity, and optimism required for the treatment of illnesses traditionally considered irremediable.

Among those who helped me with the specific task of writing this book, I wish to express my appreciation to my research secretaries, Mrs. Sara Hayes Arnaud, Mrs. Maxine W. Kumin, Mr. Bruce Herrick, Miss Alene W. Mills and Miss Eleanor Regan, for their valuable help with literature research, record research, taking of dictation, transcription of soundsciber disks and the preparation of the manuscript. I also wish to thank the artists, Mr. Ralph M. Glazier and Mr. William Dean Holt, for preparing the graphic material. I am also indebted to my office nurses, Mrs. Charlotte Doherty de George and Mrs. Helen B. Herrick, as well as Miss Jacqueline T. Turcotte, Superintendent of Nurses at Valleyhead Hospital, for valuable contributions and suggestions for the chapter on the role of the nurse. Special thanks go to Mr. Albert Grass of the Grass Instrument Company for his expert collaboration with the oscillographic study of electrical

treatment current patterns, and to Dr. David Landau for his collaboration with the respirographic study of electric coma.

I owe no small debt of gratitude to Dr. Reuben Reiter of the Reiter Instrument Company for his unfailing ingenuity and generous assistance in all problems pertaining to new developments in the use of his highly versatile electrostimulators. Without his foresight and cooperation, many of the advances in technique reported in this book would not have been made.

I should like to express my gratitude to Dr. Daniel H. Funkenstein for initiating and tutoring me in his method of testing of the autonomic nervous system, and for his availability as a highly appreciated consultant. The majority of the tests graphically reproduced in this book were carried out by him on my patients.

I am indebted to Dr. Leo R. Milner and Dr. Samuel Stearns for many valuable suggestions for the prevention and management of medical complications of shock therapy, made in the course of their ever helpful collaboration as consultants.

Last but not least, I wish to thank Dr. Walter E. Barton, Superintendent of the Boston State Hospital, Dr. Salomon Gagnon, Superintendent of Bournewood and Valleyhead Hospitals, and Dr. Charles Saltzman, Clinical Director of Bournewood Hospital, for the clinical and research opportunities they provided at their hospitals, and Dr. Jackson M. Thomas, Professor of Psychiatry at Tufts College Medical School, for the academic support and freedom enjoyed in his department.

LEO ALEXANDER

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INTRODUCTION: THE MAJOR APPROACHES TO THE TREATMENT OF MENTAL ILLNESS

There has been a great deal of progress in the treatment of mental illness during the past twenty years. Advances have been made in technique and practical results along two major approaches; by means of psychotherapeutic efforts based on increased understanding of, and resulting ability to influence, the psychodynamics of the patient; and by physical means, that is, by bringing about physical changes in the brain or the autonomic nervous system, which in turn influence the mental state profoundly.

Most of the workers utilizing these techniques have tended to limit themselves to one of these major approaches, and there have been comparatively few attempts to coordinate these rather fundamentally different lines of attack. It is understandable that the explorer will of necessity have to travel along one road, frequently a narrow one, but there is no doubt that an approach which utilizes many avenues, like an army converging upon a target, will be more effective from a practical point of view than the strict limitation to one approach, important though it is to research and development to try one approach at a time. I feel, however, that our knowledge of the treatment of mental illness has progressed sufficiently to allow a coordinated and multifactorial approach to treatment. Psychiatric therapy is still a young branch of the science and art of healing, since attempts to treat, rather than censure, mental illness are comparatively new in the history of man. Until quite recently people with mental illnesses were regarded as stigmatized by a substantial inherent weakness rendering them permanently unfit for human company, or, on the other hand, as sinners, spiritual or moral offenders against society whose illness was due to their own immorality or spiritual deficiency and who were somehow responsible for getting themselves into such a situation that society must care for them (if indeed society did). They were consequently subjected to harsh discipline, reviled and ridiculed, turned loose to fend for themselves, or, on the other hand, exorcised or preached to. The places of management or treatment were jails or religious organizations—Bedlam or Gheel.

This old dichotomy of spiritual or moral aberration on the one hand and inherent weakness on the other is still with us, and is reflected in the evolution of psychiatric theory and treatment along two major lines, the psychologic and the physical. The psychologic school, whose chief concern

is psychologic genesis and causation, first developed its thinking in experiential terms, later in terms of personality dynamics, and has most recently incorporated an increasing emphasis on social-cultural factors.

The other main line of development, that along biologic and medical-physical lines, is of more recent origin than the psychologic and actually received its major impetus from Griesinger's famous statement, "Mental diseases are diseases of the brain."² The ensuing rather narrow concern with the medical aspects of mental illness has developed a concept of the psychiatric patient as a sick man whose mental and emotional aberrations arise from and are secondary to his physical state, his disturbed body chemistry.

In the last sixty years a science of psychodynamics has gradually evolved which has for the first time made the basic dynamic psychologic factors in human personality a subject for scientific inquiry and scrutiny and has been able to integrate isolated, seemingly bizarre items into a meaningful, intelligible pattern. The consequent findings concerning the basic dynamic processes in human psychology—phenomena which the psychoanalytic school has been foremost in uncovering, formulating, and systematizing—have served as a fundament upon which further psychodynamic knowledge has been carefully built, much in the way that the basic sciences upon which medicine is founded have been evolving into a systematic body of knowledge which allows reactions to be predicted and to be influenced, alleviated, and controlled along the lines of an intelligible and workable system.

On the other hand, most of the physical means of treatment have until recently remained flashes in the pan, with a few exceptions, devoid of an intelligible causal relationship to mental states and frequently so complicated by the psychologic factors in their practical workings that they did not lend themselves to use as building stones in a science of mental disorders.

Thus stood the situation when suddenly, beginning in 1933, new tools of physical treatment became available which had one striking advantage over the older physical approaches—they worked and were effective with certain mental disorders that responded very slowly if at all to psychologic methods. By that very fact, however, although they were satisfying to the pragmatic therapist, they were vexing and confounding to the inquiring scientist because they did not make scientific sense, did not fit in with newly established theory, and therefore remained inadequate for fashioning a foundation for a new basic science of mental disorder. In other words, they remained for the time being empiric panaceas which had no visible connection with many of the gradually and laboriously developed basic principles of psychiatry.

The reactions to the development of these apparent panaceas (insulin and electric shock) were initially three-fold: first came complete emotional rejections by those more rigid protagonists of the science of psychodynamics who felt threatened to the core of their scientific identity by the inroads made by these incomprehensible panaceas. The second type of reaction, among the more flexible psychiatric scientists, was to use these panaceas as a grudgingly granted concession to a practical reality, while still adhering to the primacy of the established psychodynamic viewpoint and principles of treatment. Such men used the new tools without real

interest in them, applying them with eyes averted, as it were, and remained basically uninterested in the unknown principle behind their effectiveness, much like a surgeon who, secretly believing in naturopathy, consequently would be bound to neglect his interest in surgical technique. The third and frequent reaction was based on the practical success and seeming simplicity of the new tools and consisted of accepting them as new wonder methods that superseded all other approaches; this led to overrating the merely physical aspects in the technique, accepting it more or less as a pragmatic miracle, and so neither did these psychiatrists become interested in the further understanding and development of the new methods.

Gradually, however, a fourth group of psychiatric workers is starting to emerge and is approaching the problem from three angles: first, in a concern with the scientific investigation of the basic principles involved in the new methods with an attempt to understand them against the background of neurophysiologic knowledge, correlating them with the established facts and scientific principles of neurophysiology; second, in a study of the actual direct consequences of these methods on the patient in the light of our knowledge of the integration of higher cortical activity—for which I should like to use the term, *psychophysiology*; and third, in attempts to test and investigate the interrelations between these newly discovered psychophysiological phenomena and the body of psychodynamic knowledge. It is to these attempts that this book will be especially devoted.

Rejection of the physical treatment methods on the grounds of the academic point of view is, of course, bound to remain ineffectual. For a long time medicine was not disturbed by empiric panaceas and could develop under the strict scientific guidance of biophysics, biochemistry, bacteriology, and allied sciences. This happy state of order, however, was recently rudely disrupted by the discovery of ACTH and related endocrine substances that shook up the general principles of medicine along hitherto unpredicted lines. The fact that ACTH threatened the established principles of scientific therapeutics had a wholesome effect in reducing rigid scientific thinking among medical men and biochemists; it reestablished a searching humility.

The fact that the new physical "panaceas" in psychiatric treatment have not yet had this effect on academic psychiatry is obviously due to the fact that the shock treatments have been more difficult to integrate into the scientific principles than were the general principles of action of a new hormone. Such integration will not be possible until the general principles of the new physical therapies in psychiatry are better understood.

The complete flowing apart of the streams of development in the sciences of psychodynamics and psychotherapy on the one hand from that of the biologic or physical developments in psychiatry on the other is in some ways surprising and anachronistic because of the increasing emphasis—alas, too often lip service—given to the unity of mind and body. This divergence has been somewhat counteracted in psychosomatic medicine, where, however, the primacy of the psyche as the big brother and the soma as the little ignorant brother makes their symbiosis more acceptable to the prevailing academic psychodynamic pattern of thinking.

But even assuming the primacy of the psyche, the importance of the somatic changes occurring in mental disease has not been fully recognized

or integrated. This attitude has culminated recently in a purely interpersonal concept of psychiatry (H.S. Sullivan,^{3, 4} Frieda Fromm-Reichmann¹) and formulation in purely interpersonal terms of the disturbance of the mental patient, who is viewed not primarily as a sick person, but rather as a person who has problems and difficulties in dealing with people—that is, in his interpersonal relationships—and who unconsciously and consciously defends himself against these difficulties, thus adding new problems. The fact that the nervous system of the mentally ill patient itself functions abnormally is all too often either overlooked by the psychodynamicists or unduly emphasized by the exponents of the physical primacy in mental disease.

There is a growing body of evidence lending itself to the interpretation of mental diseases as true psychosomatic disorders of the nervous system, thus bringing about a possible synthesis of Sullivan's and Griesinger's points of view. Beyond doubt there exists an experiential stress factor in the genesis of mental disorders; there may also be an underlying predisposing somatic sensitivity; there is certainly a concomitant somatic malfunctioning which in severe cases is not remediable by psychologic therapy alone, nor is alleviation by purely physical means a substained one—relapse is inevitable unless the psychological stress has been relieved.

There is a great deal of evidence that mental disease is merely an example of a specific organ choice, namely of the central nervous system, in a psychosomatic illness. This view may be further supported by the well established observation that mental disease may alternate with other psychosomatic illnesses such as ulcerative colitis and asthma. Certain basic personality patterns of psychotic and psychosomatic patients are similar, especially the rigidity of the defenses during the healthy intervals.

It would therefore seem that in the treatment of such disorders one of the cardinal steps which holds the key to the decision as to whether or not physical treatment is to be used, is to determine whether such psychosomatic aberrations of the nervous system have yet actually occurred. If they have, and if they are of sufficient severity, physical means of restitution should be employed, with subsequent or simultaneous psychologic measures undertaken to relieve the psychologic stress and to prevent an otherwise inevitable relapse. In mild cases either approach alone may be effective. Psychologic treatment may relieve mild or incipient psychosomatic alterations; conversely, effective and quick physical relief may be sufficient to relieve psychologic stress, especially if it is recent. Physical treatment will, however, gain its optimal relieving power only if the recovery is made psychologically meaningful to the patient. In severe cases it is necessary to employ both approaches with the utmost skill. In such cases results obtained with physical means alone are too short-lived, too ephemeral; results with psychologic methods alone may be too slowly forthcoming or not obtainable at all. The effective and carefully planned combination of both methods is the basic philosophy of our approach which will be dealt with in greater detail in the following chapters.

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THE PSYCHOLOGIC NATURE OF MENTAL DISORDER

In the broadest descriptive sense, we may say mental disorder ensues when a person's perception of the world of social and objective reality becomes highly personalized in comparison to the degree of personalization by most individuals in his group, and when his emotional and intellectual reactions are so distorted by this personalization that his day-to-day functioning and relations to others become poorly adapted to his own interests in living. As the illness becomes more serious, these personalized thoughts, which no longer relate to reality, assume a repetitive autonomy.

The nature and origins of such mental disorders have occupied the best minds in the history of psychiatry; their psychologic aspects have been expressed in many terms, reflecting in general the broad sweep of contemporary thought, philosophic predilections, and often unrecognized assumptions about the nature of reality. We are most concerned, however, with those schools of thought that are still influencing psychiatry today, although, indeed, it is difficult to say if any established school of thought ever really loses all influence on subsequent thought.

THE EARLY FORMALISTIC APPROACH

Beginning, therefore, with the great psychiatric reforms in the late eighteenth and through most of the nineteenth century, we see that the formal theories then current were based on an atomistic approach to knowledge, with growing faith in objectivity through experiment and statistics, and a belief in rigid and specific causality. A great deal of effort was devoted to searching out the specific causal factors in etiology (Cameron⁵), with much reliance on exhaustive, often acute clinical description, a phenomenologic approach resorted to in the hope that resulting classifications would lead to common causes. Yet at the same time, in contrast to the statistics and classifications, there ran concurrently a rising tide of concern with the dignity and worth of the individual.

Following the humanistic trend of the times, tremendous reforms in the institutional treatment of mental patients took place in the late eighteenth century, notably under the aegis of Pinel²⁷ in France and Tuke²⁸ in England, and soon spread over most of Europe and America (Zilboorg³⁰). The enormous practical demands of the situation did not encourage investment in theory that was not immediately applicable; the psychologic aspects of mental disorder were still regarded chiefly as symptomatic, as secondary

to somatic change. To be sure, the psychologic features were not ignored and in some cases recognition of psychologic determinants existed. Esquirol,⁷ for instance, felt that emotional states might influence certain mental illnesses, although he attributed such effects more to what are today called precipitating events than to basic underlying affective patterns (Zilboorg). The emphasis, however, was on humanizing treatment, instituting hospital reforms, and on furthering psychiatric research and treatment by attention to the individual, securing case histories and keeping clinical records.

Psychology in the late eighteenth and most of the nineteenth century was still largely under the influence of philosophy. Psychologists thought in terms of faculties, neatly compartmentalized from one another, each with its intrinsic characteristics. They were far more concerned with discovering general laws than with devising theory about the non-general and deviant. When they did turn to the abnormal they did so in terms of disturbances of faculties—such as disturbances in sensation, perception, conation (see, for instance, William James' discussion¹⁷ of hallucinations under disturbances of perception, impulsive behavior under disorders of will, and the like). They tended to regard mental disorders as disturbances of intellectual functioning rather than of emotional, and assumed inherent somatic weakness of the nervous system or organic lesions as the basis.

Similarly, the influential voices in nineteenth century psychiatry spoke in terms of constitutional degeneracy, of as yet undiscovered organic lesions of the central nervous system, or of imbalance of metabolic or endocrine factors (Kraepelin²⁰). The psychologic manifestations of mental disorder were most often regarded as secondary to physical changes, and their content was considered meaningless and irrelevant, as due to chance factors. One of the main foci of interest remained in classification of mental disorders, in lumping like symptoms or details with like, in the hope of establishing discrete categories that shared common patterns of onset, course, and outcome.

The great exponent of this approach, *Emil Kraepelin*, succeeded in statistically verifying certain clinical patterns, most of which had been previously described by individual workers. His approach in its dependence on broad statistical trends necessarily neglected individual differences and atypical patterns, and in its dependence on the medical concept of onset-course-outcome it lumped together some disorders that actually had little relation to each other in terms of either etiology or process. While a stupendous work and probably a necessary step, it was unavoidably static, and as Zilboorg³⁰ points out, was rather fatalistic in that it assumed a relatively fixed course arising from relatively unalterable causes with little consideration of the reciprocal relation between the individual and the forces impinging on him that Cameron⁵ delineates so clearly.

Out of this somewhat static background of search for physical causes, there began to emerge the first systematic studies of the psychologic aspects of mental disorders, particularly of hysteria, at the Salpêtrière under Charcot⁶ and at Nancy under Bernheim.² studies later expanded and supplemented by Janet,^{18, 19} Bleuler³ and the early psychoanalysts; this systematic experimental approach stood in contrast to the previous isolated speculations of individual psychiatrists. In one sense this interest in the psychologic features was forced on these workers by the results of their

work with hypnosis, and at first it did not radically alter their assumption of the primacy of somatic genesis and the powerful influence of hereditary and constitutional weakness.

THE DEVELOPMENT OF THE GENETIC-DYNAMIC APPROACH

The studies in hypnotism and suggestion carried out at the Salpêtrière under *Charcot's* leadership followed the tradition of nineteenth century investigation and classification—for instance, of the phases of hypnosis—but for the first time these studies were not concerned only with psychotics, but also with certain reactions (hysterical) among neurotics. Moreover, while *Charcot* still considered hysteria an actual disease, he did feel that certain fixed ideas might give rise to symptoms. He found, for instance, that certain hysterical symptoms associated with traumatic accidents conformed to the patient's idea of what had gone wrong with him, rather than to any physiologic principles.

Bernheim's conception of suggestion and hypnotism was more dynamic than that of *Charcot*, who considered hypnotism in itself an abnormal manifestation (*Zilboorg*). *Bernheim*, on the contrary, regarded suggestibility as the basis of hypnotism and believed it to be a common characteristic of mankind, thus bridging the gap from abnormal to normal. He laid the groundwork for much psychoanalytic theory when, in his experiments with posthypnotic suggestion, he found that unconscious motives (i.e., motives for which the person has no memory) persist, are rationalized by the subject, and that these forgotten events may be recalled under certain conditions.

Pierre Janet went a step further than *Charcot* in attributing possible cause to purely emotional factors, though to be sure he felt this usually occurs in constitutional inferiors. He reasoned that if the normal integrative power of the mind is lessened, as by physical or emotional shock, by chronic debilitating disease, or by innate constitutional deficiencies, dissociations of thought systems or functions might occur, be mechanically split off as it were, and continue an autonomous existence. In this way he explained hysterical fugues, fixed ideas, and neurotic symptoms in general.

THE PREDOMINANTLY GENETIC APPROACH

In the work of *Breuer* and *Freud*⁴ and in the first period of *Freud's* work alone, the study of hysteria yielded its final contribution to the understanding of the role of psychologic factors. In his prolonged work with a girl with pronounced hysterical symptoms, *Breuer* discovered the almost miraculous effectiveness of catharsis or abreaction, the reliving of old traumatic situations—a finding which proved of tremendous importance in the subsequent development of the genetic approach in psychology. He concluded that certain events may be forgotten if they occur while the person is in a hypnoid state and that some people have a tendency to autohypnosis. Events and associated emotions occurring while a person is in such a hypnoid state remain outside his usual consciousness, in a way similar to *Janet's* dissociation, and although the unrecalled but disturbing experience continues to generate emotion which seeks discharge, it is cut off from normal paths of discharge, since the individual is unaware of it. Since the nervous system can stand only so much tension, however, the

blocked emotion must achieve at least distorted release, and this is the neurotic symptom.

Freud^{10, 11} at first accepted Breuer's theory, but later succeeded in explaining the occurrence of the dissociation⁹ (or repression) in more fully psychologic terms, rather than as arising from autohypnosis. He concluded that some ideas or events are not permitted awareness because of their painful nature and the conflict they awake; thus arose the first concept of repression as a defense against pain. While he at first believed that any type of emotion-laden event might be repressed, he soon found, when he changed his method from hypnosis to free association, that sexual memories and fantasies seemed most painful and were most frequently repressed and otherwise defended against and that early trauma of this nature seemed to exert a powerful and lasting effect.

From this he formed the theory that present patterns of acting and reacting are gradual outgrowths of previous patterns, altered according to the demands of successive life situations, but which may be eventually traced back to the infantile. It was here that his concept of infantile sexuality became especially important, and the influence of repression was expanded from merely dissociation of painful events to the shutting out from awareness of nonacceptable wishes, drives, and associated fantasies, which because they could not be dealt with at the patient's present level of maturity often remained exceedingly infantile. At about this time he discovered the importance of psychic reality—that a fantasy may be remembered as an event and be as important as though it actually were.

Briefly stated, fixations—that is, points of development especially invested with emotional energy—may occur in the early stages of libidinal development under certain circumstances such as constitutional predisposition, excessive frustration or oversatisfaction, trauma or excessive fantasy during a particular stage (i.e., the familiar oral, anal, phallic, with their associated attitude and behavior patterns). Such fixations necessarily involve imperfect repression since the associated affect of the wishes and fantasies still seeks some outlet (ideally such infantile wishes are effectively discharged at the appropriate period of development or are sublimated, that is, find an adequate substitute channel for gratification).

One of the basic purposes of early psychoanalytic therapy therefore became to discover the earliest hidden sources of conflict, bring the infantile wishes and fantasies to awareness, and so bring about discharge of the associated affect. While the concept of growth and gradual blending of one stage into the next, rather than sudden emergence from nowhere, is certainly in keeping with modern thought (if one overlooks for a moment theories of creative emergence), the desire to trace back to the earliest model implicit in the genetic approach—as well as in much Freudian thinking—in a sense represents a deterministic hang-over of the search for a specific cause that once found and eradicated would eradicate its effect.

In discussing Freudian theory in any full way—something we cannot undertake in this context—it is necessary to do a great deal of backtracking to compare early and later viewpoints and theory. The system developed slowly, was at first much influenced by Freud's work with hysterics, shifted its points of emphasis, and finally became increasingly concerned with the

active defensive functions of the personality, the ego operations, and the conflict potentialities among the conceptual triumvirate of functions, id, ego, and superego forces. This still does not begin to encompass the later divergent developments based on psychoanalytic foundation.

While the unveiling of all infantile fantasies is no longer the first aim of psychoanalysis, which has now become much more concerned with exploring ego defense operations, the genetic emphasis is still important in psychoanalysis and is an important concept in the handling of the transference situation, which is seen as a way of working through (though not necessarily verbally expressing) old conflicts, fantasies, wishes, and repetition compulsions or stereotyped interpersonal reactions in the present controlled reality situation. In contrast to this shift, Freud's original separation of the psychoneuroses as those whose genesis lay in the infantile life, from the "actual" neuroses whose origin tended to lie more in present circumstances (usually physical) has now to a large extent faded away.

Perhaps the most significant changes in Freudian theory came with the introduction and expansion of the structural approach, that is, with the abstracting of three broad systems of personality function, termed id, ego, and superego. The earlier emphasis, no doubt largely influenced by Freud's work with hysterics, was chiefly on those functions of the personality represented by id and superego. Subsequently the executive, adjustive, coordinating, integrative functions of the personality were given much more weight, and this trend greatly influenced subsequent theory, including much modification of the theory of the power of instinctual development on character formation. With Anna Freud's work⁹ came the realization that character formation is strongly influenced by the patterns of defense resorted to by the ego. The original single source of emotional energy—libido or the underlying drive for organ pleasure—was expanded into a dual source, with aggressive mastery drives as the second great system of emotional energy; and there has been considerable speculation lately about the source of nonsexual, nonaggressive psychic energy and debate about whether it is intrinsic or arises from sublimated sexual or aggressive energies.

The recognition of the exceedingly complex development of personality, neurotic or otherwise, in which so many diverse factors play a part, interacting among themselves and having different effects on different people and on the same person at different times, has led to much less emphasis in therapy on early beginnings and to more concern with the present situation and with what Cameron⁵ calls that point in an event sequence where we can interfere constructively to shift the direction of the sequence.

THE PREDOMINANTLY DYNAMIC APPROACH

The event-sequence modern dynamic psychiatry is most concerned with is that dealing with the production and perpetuation of conflict, the nature of the defensive operations resorted to, the role of anxiety and guilt in the dynamic functioning personality constellations, and the myriad interrelationships. Conflict is now generally recognized as the main basis of neurosis and of at least the psychologic aspects of psychosis, with anxiety as the energizing force behind defensive operations, and symptoms as manifestations of the conflict which may serve various functions or may

represent crippled function. With this comes the present conception of the choice of neurosis being determined by the type of defensive operation the ego resorts to, in contrast to the earlier Freudian theory of origin according to different types of early trauma.

Just as the present-day dynamic approach, while still related to the genetic, has changed its emphasis to the contemporary, it has also shifted its emphasis from the biologic-instinctual to the cultural-social under the tremendous influence of the cultural anthropologists and the demonstrated differences between certain patterns of development in the Vienna of Freud's day and in other cultural groups. In line with these changing conceptions, Frieda Fromm-Reichmann¹² remarks that in our present social setting the emotions most frequently leading to severe conflict and requiring the mobilization of strong defenses are not so much those concerned with sexual impulses as those concerned with hostility and aggression, since, in our democratic society, open expression of hostility against others is one of our most strongly tabooed reactions.

Much of the work and thought of the current "neo-Freudians" reflects these shifting emphases in the concern with the present life situation, the increasing doubt about the innateness of certain stages of psychosexual development, and the concern with interpersonal relationships as the source of everything psychologically meaningful to the individual. *Horney*,^{14, 15, 16} for example, describes the specific content of neuroses that arises in our cultural setting with its great and conflicting demands and with its socially accepted ways of dealing with conflict. While she retains a genetic orientation in terms of the early experiences that make fertile soil for the development of neurosis, her main concern is with the present life situation of the individual. She expresses strong doubt about the innateness of certain trends—e.g., the oedipus complex, penis envy—instead considering them products of the interpersonal relationships arising in our particular social-cultural setting. She regards many neurotic symptoms not so much as distorted gratifications but as compulsive patterns aimed at safety. This emphasis on security operations rather than libidinal gratification and on the importance of interpersonal relationships and their distortion in neurosis is the common mark of the Washington school of psychiatry that was so greatly influenced by Harry Stack Sullivan.

Although the psychoanalytic approach has been exceedingly fruitful in research, understanding, and therapy, to say nothing of its jolting vivifying effect on other schools, it was not the only constructive reaction to the determinism, fatalism, and static formalism of the nineteenth century. The seeds of change had been sown wide; the philosophy of pragmatism was spreading; the importance of individual subjective states recognized; and the relationship between each idiosyncratic individual and the factors in his actual life situation, the natural, social, and cultural setting, began to come into focus—human nature was seen increasingly as relatively plastic rather than as biologically predetermined.

These changing points of view were reflected in all important psychiatric systems of the day, as well as in related fields; many of the trends and discoveries were supported—or corrected—by evidence from quite independent sources. All shared certain similarities in philosophy and fundamental assumptions about human functioning, although each elected to emphasize certain aspects and often chose to point up their differences in

wording and degree of specificity, neglecting much of their basic similarity. The psychoanalytic approach, for instance, at first chose to disregard to a large extent the physiologic accompaniments and possible basic mechanisms in psychic reactions, perhaps in a compensatory swing from the previous fruitless overemphasis, and even though Freud continued to maintain his biologic orientation, assuming, however, that the intimate interconnections could not yet be worked out in the present state of our knowledge. (This has, incidentally, been corrected in certain branches of subsequent psychoanalytic development, particularly in the work of the Chicago school on psychosomatics.) Other workers, on the contrary, chose to make more out of physiologic or other aspects of this tremendously complex system of interrelationships.

How these historical trends were carried on in other schools of psychiatry will become evident in considering the work of three prominent and influential figures in modern American psychiatry—McDougall, Meyer, and Myerson. (It is obvious, of course, that none of these trends remained uninfluenced by the ideas of the others.)

THE PSYCHOBIOLOGIC APPROACH

William McDougall,²¹ although he has fallen out of fashion because of his instinct theories, was in some respects an entirely dynamic psychologist. His constant emphasis on teleology, on hormic psychology as he called it, on purpose (conscious or not) behind every action is quite fully accepted today by modern psychoanalytic psychology which sees purpose in every act no matter how inconsequential it may seem. He was entirely unconvinced of the necessity of somatic genesis in severe mental disorders, pointing out that structural neuronal changes and changes in endocrine function may be as reasonably explained as the effect of function on structure as vice versa: "The demonstration of organic changes in a case of mental disease of some standing does not by any means prove the disease to be of organic, rather than functional origin. Only the demonstration that changes of one order precede in time all changes of the other order could suffice to establish the primacy of the functional, or of the structural, derangement" (p. 44)—a point we might do well to remember today.

Although McDougall recognized the existence of subconscious purposes and believed that conflict is basic to "a large proportion" of mental disorders, he did not fully accept Freudian doctrine, in particular its early tendency to monocausality and phylogenetic speculations. Since he considered every tendency, whether or not conscious, as purposive, striving for a goal, he saw the primary conflicts not only as those between the interests of the individual and those of the species and social community but also as between various instinctual tendencies all striving for maximal development, a perpetual rivalry that normally resulted in reciprocal checking and harmony, but led to inner conflict if one system of tendencies became too strong.

McDougall's orientation was functional and dynamic, and at the same time he was interested in theoretical formulations. His instinct theories, however, have not proved particularly fertile—although, to be sure, Murray²⁴ has revamped certain of his concepts quite profitably.

Adolf Meyer,^{22, 23} with his tremendous influence on American psychiatry, represents a somewhat different reaction from the nineteenth century

brand of psychiatric thinking. He did not adhere to any theory in particular, but instead emphasized the pluralism of cause and was disinclined to specificity, in contrast, for instance, to the phase of psychoanalytic theory then in force. In a sense he was something of a psychiatric agnostic—holding that since most of the basic causes and relationships in human functioning were unknown and for the present unknowable, the only feasible approach was a pragmatic, empiric one that took into account the actual functioning of the unique living organism in its unique life setting. He felt that attention should be directed to those factors that could be seen at work. This he called the “common-sense approach,” and it was indeed a breath of fresh air after the stifling atomistic rationalistic thinking that preceded him. He advocated the study of “facts” for what they actually mean in the life of the specific individual. For this he proposed certain generalizations of type of psychopathologic reaction, rather than classification by disease entity (since he was not convinced mental disorders were diseases). He was interested not in absolute measurement, but in rhythms and orders in the functioning, activity and rest phases of the human organism in its totality; as his term “psychobiology” implies, he did not think it possible to consider psychic aspects apart from biologic.

THE VITALISTIC APPROACH

Another outstanding representative of the pragmatic, multifactorial nonspecific approach in American psychiatry was *Abraham Myerson*.^{25, 26} His thinking reflected current trends in concern with cultural factors, in his pragmatic approach, his belief in the identity of mind and body, and his concept of vitalism. He was, on the whole, content to be descriptive and pragmatic, and like Meyer skeptical of specific answers and theory.

Myerson's thought was firmly embedded in what he called the organistic approach; as he remarked: “From the highest to the lowest mental function, there is a grounding in organic structure which makes mind and body fundamentally and pragmatically one” (1927). Like Meyer he thought one must consider the organism as a whole “as a place where environmental forces of all kinds enter and are reacted to” (1927).

Myerson was much concerned with social determinants of personality and with the basic conflict between the demands of social convention and the fundamental needs and drives of the human animal; his emphasis on this is in remarkable accord with the modern dynamic orientation of psychoanalysis with whose genetic theories he was in disagreement. He felt that social psychopathology led to much individual psychopathology by the strains society sets up against natural inclinations, the frustrations it imposes, and the conflict it perpetuates between biologic drive and social injunction. In speaking of the demands of civilization he remarked, “The organization of activity for the future means creating a form of social ambivalence, the denial of the present moment. This, I think, is the chief strain in man's life, a strain easily endured by a fortunate few, troublesome and disturbing to many, and tragic to others” (1950, p. 227).

Myerson's most interesting contribution, perhaps, was his emphasis on vitalism, on strength of appetite and desire as the fundamental requirement, the *sine qua non* of mental health. This zest, this “appetite for living” that encompassed the “vast innate range of desire,” was, he felt, quite nonrational and primitive, bearing no relation to moral code, and

had as its source feelings and sensations arising from within the body. "Awareness of these impulses is usually only in the background of consciousness, but is intimately linked up with the state of well-being. To the individual his own inner state is of more intimate and intricate importance than is the rest of the universe" (p. 217, 1950).

Myerson's concept represents the clinical epitome of Bergson's *élan vital*, that free creative energy Bergson believed to be the principle behind cosmic and individual life processes (Bergson¹). William James remarks: "The basis of our personality . . . is that feeling of our vitality which, because it is so perpetually present, remains in the background of our consciousness," p. 207, 1948). It is certainly closely related to Jung's concept of libido, as well as to Freud's, and to what Weiss²⁹ and Federn⁸ name "medial cathexis." McDougall also speaks of "vital energy." What is important is that Myerson chose to elect it to the role of primary significance in the maintenance of mental health.

THE APPROACH IN TERMS OF HARMONY

An extremely interesting philosophic view of mental functioning was proposed by *Gurdjieff*,¹³ who believed that mental health is dependent on the harmonious activity of the various components of personality, on "the totality of automatic functioning." Illness to him was disharmonization; psychopathology arises when one reacts "with only one part of one's entire presence." Implicit in his view is that proper treatment of a condition that consists of dominant disordered reaction by only one component lies in stimulating the entire healthy but dormant background of the personality to action.

In a sense this view may represent a philosophic rationale for various forms of stimulative treatment—the practical epitome of which may be Myerson's "total push" regimen. In a way Gurdjieff's approach entails a vitalism that gives more attention to the workings of the specific parts. In an illuminating metaphor, he distinguishes "the owner of the carriage" (the essential purposive self) from "the coachman" (the intellectual, directive conative self), "the horse" (the instinctual driving forces), and "the carriage" (the body). The true self must induce the coachman to work loyally for the higher purposes that only the real self possesses, for the coachman might stray away and be tempted by more frivolous impulses.

I interpret Gurdjieff to mean that in mental disease it is usually a relatively small segment of the personality that goes wrong, while the total vast richness of the personality, apart from this segment, remains unimpaired, though often latent. The aim of treatment lies in stimulating the remaining rich normal reservoir so that it may overcome the disordered part and draw it into normal functioning—in other words, to stimulate the normal resources to overcome and normalize the small part that has gone wrong. Otherwise, so to speak, a small minority will terrorize the majority.

After this survey of divergent approaches it becomes apparent that at present we stand at a crossroads where different approaches meet and infiltrate each other; that many of the apparent differences are semantic and emotional or are based on a too narrow view that excludes important segments of data. All these approaches shade into one another at various points, and, with the partial exception of the work of the great pioneers, seem largely determined by historical trends and development in thought.

Concern with etiology now appears to be passing into concern with process, with event sequence. It is apparent that the two wings of modern dynamic psychiatry—the psychoanalytic and the pragmatic-holistic—bear somewhat different emphases, the former concerned with etiology and process (based on observation) and leading to the development of a large bulk of the genetic-dynamic-cultural theory, but which occasionally in the application becomes theory bound and results in a narrowing view of the data and their meanings; while the empiric-pragmatic approach has developed consistent theory far less and has depended largely on the truth it imputes to its basic assumptions and knowledge for dealing with psychic phenomena in a holistic way. This has in some ways led to a more expansive view and to more concentration on present patterns of function, continued interest in somatic aspects, and greater readiness to accept and interpret new psychologic and physical approaches.

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SOMATOPSYCHIC BACKGROUND AND PSYCHOSOMATIC NATURE OF MENTAL DISEASE

The oneness, the essential unity, of the human organism is perhaps nowhere more evident than in psychiatric disorders, for here the integration of the various levels of organization, function, and regulation that make up the extreme complexity of this living system stands out in bold and striking relief. Nowhere does the ancient dichotomy between mind and body seem more artificial.

Nevertheless, the casts of thought arising from that contrived separation are deeply embedded; it is a division ingrained in our historical-cultural tradition, a product of centuries of philosophic and theological thought and of man's sense of his own importance and superiority to other creatures in the animal kingdom. It has been further perpetuated because of the ease with which a superficial distinction between mind and body can be made and because two quite different methodologic approaches—psychology and physiology—are easily formed and lead to knowledge of apparently different orders, although actually their findings relate to different levels of function. While medically and biologically this separation has proved convenient and workable in the past, this is no longer the case; the concretization of the abstract dichotomy is proving a stumbling block to further understanding of human beings.

Today the interaction and, essentially, the identity between mind and body is widely accepted in theory, but a balanced picture of the process is still in the making. The most obvious and perhaps oversimplified statement of the relation is that influences and forces impinge on man from two directions and in a constantly interacting way—from the outside through his sensations and perceptions, what he makes of men and how he reacts to them in the light of his unique personality and constellation; and from the inside in sensations and perceptions of his bodily state, in his sense of physical well being, his energy level, the effects on his nervous system of metabolic balances, toxins, pathology, influences of structural relations and changes, endocrine and biochemical relations and the like—in other words, everything that affects the internal economy of this relatively self-contained, self-regulating, creative unit.

Two sources of influence may thus be abstracted, rather artificially, but the being reacting to them is one. We know that “mental” reactions eliciting emotions and attitudes may have marked effects on the functioning and structures of the body and that physiologic changes may have

marked effects on mentation. The interactions are constant and result in perpetually changing, shifting balances.

Nevertheless, the initiating or predominating influence in psychiatric disorder may come from the basic somatic level—in terms of constitution or physical change—and reverberate upward to influence higher levels of function and regulation; or the disorder may be primarily of a psychogenic nature, arising out of the patient's perception of his experiences and needs—which, in a sense at least, is a function of the highest integrative phase of the nervous system and which may in turn set up varying degrees of disturbance among lower levels of function. In the preceding chapter we investigated the question of the extent to which mental disease is anchored in the mind. In this chapter we shall discuss to what extent it is anchored in the body and to what extent it may change the functions and at times the structures of the body.

HEREDITY

The oldest clue to a physical anchoring of mental disease has been thought to lie in the positive evidence of heredity. This, however, has turned out to be far from the simple problem it was once believed; it is an issue beclouded, not to say bedeviled, by many factors. One of the most important considerations in any study of genetic transmission of mental disorder is whether or under what circumstances familial association and heredity may be equated, to differentiate between what is inherited biologically and what is ingrained in the constitutional make-up by early experience. But first a word about the actual statistical studies on the incidence of mental disease in family groups.

Many of the early studies that sought to establish evidence of "tainted heredity" have been subsequently found seriously deficient in statistical methodology and in selection of cases and controls. Moreover, any evidence of what was then considered mental degeneration was considered positive evidence of constitutional neuropathic make-up, the theory being that specific forms of mental disorder might well arise from a general constitutional deficiency of the nervous system—a theory not without merit, except that many characteristics were included that seem to have little direct relation to the quality of the nervous system or are now established as of social or other extrinsic origin, including tuberculosis, pauperism, and bodily deformity (Myerson et al.⁴⁰). Such polymorphic inheritance may arise from an innate deficiency of nervous integrative and regulatory powers, or it may be more influenced by poor environment and poor early conditioning and interpersonal experiences.

In spite of the rather inadequate statistical methods employed and the dearth of adequate control studies, there seems to be fairly conclusive evidence that there is a higher incidence of schizophrenia in some families than in the general population. There is, however, no clear-cut evidence of Mendelian inheritance, dominant or recessive. There appears to be a clearer tendency for manic-depressive psychosis to be associated with family groups, especially when cyclothymic personalities within the family are also included, but the relation is not to the point of Mendelian dominance. (Myerson et al.⁴⁰)

Studies of monozygotic and dizygotic twins present much stronger evidence of a genetic substratum predisposing to manic-depressive and schizo-

phrenic psychoses. Such comparisons of twins, reared together or apart, give rather straightforward evidence of a significantly higher incidence of similar psychoses among identical twins than among fraternal twins (Kallmann²⁹; Myerson et al.⁴⁰). From these studies it appears, however, that predisposing heredity is not an imperative to development of mental disorder, even in similar environments, but that it may be a powerful potential. It is interesting to note that while familial association is somewhat clearer in manic-depressives than in schizophrenics, nevertheless, according to studies of twins, environmental influences may be more important in evoking a manic-depressive psychosis than they are in evoking a schizophrenic one—i.e., among monozygotic twins the incidence of psychosis in both twins was higher among schizophrenics than manic-depressives (Myerson et al.⁴⁰).

On the other side of the picture, note must be made of the fact that either a schizophrenic or a manic-depressive episode may occur in individuals whose pedigree gives no significant evidence of mental disease.

In dealing with this far from distinct picture of the genetic inheritance of schizophrenia, Kallmann²⁹ proposes the theory that susceptibility to schizophrenia is based on a specific unitary, probably recessive hereditary factor. Such specific predisposition, however, is counterbalanced in varying degrees by constitutional capacity to resist the disorder, based on various genetic but nonspecific factors which contribute to the formation of what he calls a "mesodermal defense mechanism" against developing schizophrenia; that is, he relates this power to resist quite directly to body type. He has noted, for instance, that the greatest deterioration seems to occur in schizophrenics with asthenic builds; and if only one twin develops schizophrenia, the nonpsychotic twin is found to be larger and stronger (as reported in Bellak⁶).

The reasons for this rather diffuse, statistically unconvincing picture by no means lie merely in the difficulty of establishing hereditary influences upon the nervous system, since such influences have been well documented in some disorders such as Huntington's chorea, certain types of mental deficiency, Leber's disease, and others. Rather the difficulty seems to arise from trying to ascribe a unitary, more or less specific type of inheritance to mental disorders.

It is now apparent that the etiology of schizophrenia is diverse and multifactorial. Schizophrenic reactions may arise in response to a variety of stress situations, including infectious diseases and organic changes in the nervous system (Bellak⁶), although of course it may be argued such changes may merely act as precipitating factors. There does, moreover, seem to be a type of schizophrenia with a characteristic pattern that is rather closely associated with high familial incidence (Weinberg and Lobstein,⁴⁹ Bellak⁶). Bellak's argument for considering schizophrenia a type of reaction with nonspecific etiology, that may, in fact, arise at any point on a theoretical continuum from purely somatic and/or genetic to purely psychic influences, seems well founded.

Actually, inheritance of predisposing tendencies to mental disease is probably not unitary, but rather configurational, arising from the combination and interaction of many factors at a level much lower than the psychic—the final picture being the result of multiple interacting factors and certain "outside" conditions of stress and/or demand. It is as though

these mental disorders were related to an unknown factor complex, which in turn bore some relation to familial groups. The nature of such intermediary factors can only be speculated about at this stage—whether, for instance, it is on the basis of innate energy potential as some workers suggest (Halperin and Guensberg²⁰), of more obscure metabolic or enzyme factors that contribute to actual nerve-cell functioning as Hoskins²⁵ mentions, of hypothalamic dysfunction as others have suggested, or whether all these things are merely aspects of some more fundamental change.

The emphasis in modern genetics is to regard heredity as a potential only, that may be evoked or left dormant by the environment. The action of the gene is not unitary, but rather it blends with and is modified by the action of other genes. Halperin and Guensberg²⁰ comment in this connection: "The point of reference in human genetics is the gene, but geneticists would discourage the popular practice of relating a specific disease or trait to the isolated action of a particular gene. Rather, it is emphasized that perhaps the entire complex of genes is involved and that a trait or character is merely associated with the gene that exerts the most marked effect and that its expression is dependent upon the peculiar environmental influences in which the gene happens to be active."

CONSTITUTION AND BODY STATE

Somatype. While certain extreme types of body build have long been recognized, together with their affinity for certain diseases, Kretschmer³³ was one of the first psychiatrists to make systematic observations of relationships among body build, temperament, and the prevalence of certain body types in certain mental illnesses. He established a relation between manic-depressive psychosis and pyknic build, between dementia praecox and asthenic-leptosome build, and between paranoid and epileptic states and athletic build, relationships that have since been essentially confirmed.

Sheldon⁴⁶ took up where Kretschmer left off, adding to the concept of typologies the concept of continuities, that is, of one type shading into another in the general population, with each type as an extreme expression of one of the three main characteristics of every body build. While his types correspond to Kretschmer's (pyknic = endomorph, athletic = mesomorph, leptosome = ectomorph), he considers in addition the relative balance of the various components in each individual, something Kretschmer did only in his category of dysplasia. Sheldon offered theoretical justification for the emergence of the three components by relating them to the embryologic layer from which the characteristic developed—endoderm (visceral), mesoderm (somatic-muscular), ectoderm (nervous). He introduced operational definitions of the various components, together with standardized procedures and measurements, and he has attempted by extensive sampling to establish norms for the general population.

The relation of body build to heredity is fairly well established as quite direct, but as influenced by the extent to which the environment allows development of the innate potentials. The extent of the relationships between temperament and body build and between certain mental and psychosomatic disorders and body build is still being explored, but the evidence so far seems to support rather high positive correlations.

Betz⁷ (reported in Bellak), for instance, in a careful survey among female schizophrenics, found apparent relationships between body type

and premorbid personality, and course of the disease. The majority of the schizophrenics she measured were of asthenic build, they showed greater variability in physical measurement than the general population, and they tended to be smaller. Asthenics tended to have had schizoid personalities before developing frank psychosis; they usually developed a psychotic episode before they were thirty; and they tended to deteriorate more often than other body builds. Patients with pyknic and intermediate body builds were less frequent among the schizophrenic population and tended to show different premorbid personalities and different courses of disease than did asthenics. Those of intermediate body build (which most closely corresponds to Kretschmer's athletic and Sheldon's mesomorph) were found least often to have had premorbid schizoid personalities and least often deteriorated.

Kline and Tenney³¹ report on the relation found in schizophrenia between certain diagnostic subtypes and somatotype and between somatotype and prognosis. Their study seems especially valuable because their methods minimize the subjective elements of judgment. Serial admissions to a Veterans Administration Hospital were routinely subjected to the standard photographic procedure for determining somatotype, regardless of diagnostic category on admission (including nonpsychiatric cases). The photographs were then rated and assigned somatotypes by Dr. W. H. Sheldon, who had no other knowledge of the patient. The hospital staff, however, was not informed of the somatotype rating of individual patients and the usual hospital course was in no way influenced by them. Two years after admission of the first patient and six months after admission of the last in the series, disposition and diagnosis of the patients were reviewed in relation to somatotype, and certain relationships of high statistical significance were established.

There was found to be a significant positive correlation between good prognosis and mesomorphy as the dominant body type; while prognosis appeared poor among patients with high endomorphic component and among those who received either quite high or quite low cumulative ratings on the three components—i.e., who were either "overendowed or underendowed" constitutionally. It was also found that mesomorphs tend to the paranoid subtype of schizophrenia and very seldom become hebephrenic; while ectomorphs seldom become paranoid but instead dominate the hebephrenic group. (No significant correlation between somatotype and other schizophrenic subgroups was found.) The relationship between somatotype and prognosis proved more telling than that between somatotype and diagnosis, and the two relationships were found to be independent of each group. The authors point out that the preselection that existed in the group by virtue of their being veterans probably means that a good many endomorphic and ectomorphic extremes had already been eliminated in that they had broken down before being admitted to military service.

The ability of the person with dominantly mesomorphic components to resist schizophrenic breakdown and to recover from it seems quite strikingly illustrated in this study and confirms other findings and clinical impressions, including Kallmann's. It would seem to have many implications that may be regarded both physiologically and psychologically. Kline and Tenney³¹ remark that evidence from this and other studies yet to be published indicates that "many physiological responses—and ultimately

the biochemical and biophysical relations upon which they rest—vary in direct relationship with somatotype.” The superior physical endowment of the mesomorph from the point of view of strength and endurance certainly seems pertinent in relation to his relative resistiveness to schizophrenia. That this type should tend to develop the paranoid form of schizophrenia is interesting—this is the form Hoskins²⁵ declares most nearly retains psychosomatic normality; psychologically this is a form of the illness in which the patient believes he has an objective foe, something he can fight—this reaction in the person physically best equipped to fight.

The relationship of body build to developing or resisting various forms of psychopathology is most provocative, particularly the apparent affinity between certain specific types of mental and psychosomatic disorders and certain body types. It is obvious, however, that the relationship is far from a direct one in most cases, since many people of similar builds do not develop these illnesses nor do they necessarily have attenuated trends toward them. And the questions still remain: is it the type of metabolism or biochemical relationships associated with given body builds that provides the physiologic substratum for defective functioning? Or is the relation due rather to the sort of reactions to life experiences that are most suitable to these people by virtue of their constitutional equipment? Or is it the constitutional equipment itself that predisposes the person to certain sensitivities to environmental influences and to certain types of experience?

As a concrete example, is it the possible superior physiologic functioning of the mesomorph compared to the ectomorph that provides him with greater psychosomatic resistiveness to developing schizophrenia or once having developed it to recover from it? Or could it be his greater capacity to fight—the type of reaction he finds most rewarding with his particular equipment? Or is it that people of athletic build are more favored in our society, are more accepted and admired, find greater opportunity for satisfaction and security?

The same question, *mutatis mutandis*, may be applied to the other extremes of body type—for instance, the greater nervous sensitivity of the ectomorph to outer stimulation that has been pointed out by Sheldon,⁴⁷ Draper⁸ and others. From the point of view of character development, it is worth considering whether certain stages of development offer greater satisfactions or securities to certain constitutional extremes and whether a consequent immaturity or overreliance on certain types of satisfaction or security associated with various developmental phases may not contribute significantly both to type of mental disorder and to prognosis—e.g., as has been suggested by others, whether the endomorph finds greatest satisfaction at the level of oral-gastrointestinal modes, whether the ectomorph by virtue of his greater nervous sensitivity seeks shelter and respite from the hurly-burly everyday world, whether the mesomorph finds greatest satisfaction in the active aggressive approach most approved by contemporary society?

This concept of one particularly meaningful role of the physical constitution applies not only to schizophrenia, but to certain neurotic developments as well. In fact, it is clinically sometimes more obvious in neurotic developments. I have first been impressed with this possible meaning of physical endowment in my work with children, mostly boys, suffering

from specific reading disability. These children almost invariably develop superimposed neuroses or behavior problems, with one notable exception: namely, when they manage to become good, or better still outstanding athletes. This happy circumstance obviously assures them of adequate social recognition and frequently leadership. I have used encouragement in this direction in my therapeutic work with them. In this connection it is of interest that children with specific reading disability frequently have a natural gift for the development of athletic prowess. Hughes and his co-workers²⁶ have adduced electroencephalographic evidence that this may be due to a preponderance of the development of the cerebral projection systems over the association systems. In the case of a girl with specific reading disability whom I saw recently, a neurotic upheaval was initiated by her mother's ill advised attempt to make her improve her reading by making her give up her rhythmic and acrobatic dancing classes in which she excelled. Restoration of this important source of self-respect and recognition, in addition to other changes of the environment in the direction of permissiveness, combined, of course, with the initiation of special remedial teaching for her reading disability, relieved the situation.

Early Influences by Physical and Emotional Environment. In discussing constitutional predisposition to mental disorder, evidence of early constitutional conditioning should be considered, especially from the point of view of its relation to familial associations in various disorders in distinction to genetic predisposition. An interesting case illustrating the possibility of prenatal influences on constitutional make-up was that of a woman whose pregnancy had to be interrupted because of a severe agitated depression. When Dr. R. Ashbel⁵ examined the placenta of this woman, she found an extraordinarily high proportion of active carbonyl groups in it.

If this finding should be borne out in larger-scale studies, it is conceivable that this type of emotionally agitated mother may actually bombard the growing fetus with adrenal products. The effects of such a bombardment on the highly plastic system of developing cells can be only speculated upon, but it may well be that certain cells in the fetus are specifically influenced functionally by this abnormal environment in such a way as to have a marked effect on subsequent sensitivity and adjustments to stress. Phyllis Greenacre¹⁹ has also done very interesting work on possible prenatal and birth influences of a physical order that may predispose the individual to the development of anxiety of varying degrees of intensity on relatively mild provocation.

The instability of the incompletely developed nervous system of the infant, which extends over the first few years of life and is especially evident in the first three months after birth, renders the child particularly susceptible to certain environmental influences, especially prolonged or intense ones, that in the early, still precarious stage of nervous and endocrine development may have a marked effect on the patterning and conditioning of ensuing integrations, both physiologic and emotional. Ribble^{44, 45} has shown that if certain conditions basic to the healthy development of the infant's nervous system and his first psychomotor integrations are not met, the consequences may be quite grave. Observations of children whose first two or three years were spent in a highly impersonal institutional environment support her thesis and indicate that such a lack of

early physical and emotional stimulation may result in a basic impairment of the ability to form affective ties with others and to operate in an integrated, effective manner.

Body State. Alteration in the normal conditions of the body state—whether due to deficiency, depletion, toxins, infection, or the like—may bring about profound changes in the mental and emotional state of the affected individual and may at times present a clinical picture similar to that of the functional psychoses. On the whole, the various recognized categories of these so-called exogenous mental disorders have a specific etiology and often present a specific clinical picture, although the content tends to reflect the unique personality dynamics of the individual. A few of these conditions are Kleist's³⁰ postoperative psychosis, the alcoholic psychoses such as Korsakoff's³² psychosis and delirium tremens, psychosis resulting from tetraethyl-lead poisoning, and psychopathologic symptoms associated with various vitamin deficiency states, fatigue, dysentery, infections, and structural changes in the nervous, circulatory, and endocrine systems.

PSYCHOSOMATIC CONSIDERATIONS

Certain somatic anomalies are found rather consistently among mentally disturbed patients. These abnormalities seem to reflect broad trends in dysfunction consisting chiefly of shifts in physiologic balances, reduced or enhanced responsiveness, and disturbances of homeostasis and adaptability, together with their effects on the efficiency and stability of the organism.

Certain of these disturbances are more prominent in some disorders than in others. As might be expected, the imbalances are more severe and pervasive in psychotics than in most neurotics. While the majority of physiologic studies have been concerned with exploring the somatic reactions of schizophrenics, there is evidence that similar though not necessarily identical imbalances are present in all the functional psychoses.

Neurotics, aside from those with clearly recognized psychosomatic disorders (that is, those who have somatized some aspect of their conflicts), appear to show their chief somatic abnormalities in their prolonged and intensified anxiety reactions in which homeostatic readjustment of the various somatic components is quite impaired (Malmo et al.³⁶); (Funkenstein et al.⁹⁻¹⁶). The neurotics' biochemical responses to stress have been found to be essentially like those of the normal, whereas those of the schizophrenic are consistently different in certain details (Hoagland et al.^{22, 23}; Pincus et al.^{41, 42, 43}). On the other hand, Altschule^{2, 3} and Gildea¹⁸ have been unable to agree with Hoagland and fail to find an essential difference between the schizophrenic and the normal in these respects.

In general, the findings on physiologic functioning in schizophrenics indicate defective homeostasis and sluggish responsiveness in many bodily sub-systems—circulatory, equilibratory, glandular—as well as evidence of defective metabolic processes (Hoskins²⁵; Bellak⁶). In many patients, however, the "only consistent difference from normal lies in the greater variability of values for almost any factor investigated."⁶

There have been rather extensive observations of the response of schizophrenic patients to stress of both physiologic and psychic nature^{4, 6, 12, 15, 17, 21, 22, 23, 25, 36, 37, 41, 42, 43} with the consistent finding of decreased adrenocortical responsiveness, in spite of evidence that the adrenal cortex is

capable of increased function in these patients; in the minority of schizophrenic patients who did evidence apparently normal adrenocortical responsiveness, the quantitative ratios of the components of the response differed from the normal (Pincus and Hoagland⁴³). This type of hypoadrenalism is related to the deficient potassium metabolism found in many schizophrenics—and potassium is a very important electrolyte contributing to the excitability of the nervous system and to its ability to generate and propagate electrical activity (Pincus and Hoagland).

Hoagland and his associates²³ found similar states of adrenocortical unresponsiveness in patients suffering from involutional depressions. As the depression lifted, however, under the influence of electroshock treatment, the responsiveness of the adrenal cortex increased.

Such evidence of concomitant physiologic impairment has led some workers in this area to formulate such hypotheses as the following: "It may be that [such] endocrine deficiencies . . . make some persons more vulnerable to the stresses of living than others. Such persons may never become psychotic if their lives present few problems, but under more severe environmental and personalized stresses their endocrine defects may result in brain malfunction with consequent psychotic disturbances." (Pincus and Hoagland,⁴³ p. 650.)

Similarly Hoskins concludes from his examination of the biologic aspects of schizophrenia that: "The schizophrenic psychosis is marked by numerous defects of adaptive efficiency, leading to inadequate and uneconomical organic responses to changing stimuli. . . . It would seem that the prodigality of effort required for organic adaptation leaves the patient with but inadequate energy for successful adaptation in the social field." (Hoskins,²⁵ p. 159).

The Funkenstein Test. Certain more subtle changes, and incidentally more easily detectable ones than those just described, were found by Funkenstein and his coworkers⁹⁻¹⁶ in the patient's psychic and autonomic reaction to two drugs with opposing effects on the autonomic nervous system—*epinephrine*, which with its adrenergic effect may serve as a measure of sympathetic reactivity, and *mecholyl* (acetyl-beta-methylcholine, methacholine), which with its cholinergic effect may serve as a measure of parasympathetic reactivity, or rather as a counterbalance of sympathetic reactivity. In the test devised by Funkenstein and his coworkers for this purpose, he uses the systolic blood pressure as a measure of autonomic reactivity to each drug after the basal blood pressure level has been established, determining the intensity of response and the ability to reestablish homeostasis.^{10, 11, 12}

He found that while normal controls generally show a moderate response to the drugs and reestablish homeostasis rapidly, mental patients usually show either exaggerated or weak response. "often with deficient capacity to reestablish homeostasis." Moreover, some patients reported acute feelings of anxiety as soon as the action of either or both of the drugs began to take place—this reaction was most frequent in anxiety neurotics. Funkenstein and his coworkers believe it may be explained on the following basis: "In the individual who suffers from anxiety, awakening the somatic manifestations in intense form recalls the subjective components which are closely associated thereto in experience and in conditioning." Funkenstein and his coworkers note that anxiety in the clinical

sense may be expressed in different patients by different physiologic manifestations, some depending mainly on sympathetic pathways, some mainly on parasympathetic, some on both, while others—apparently equally

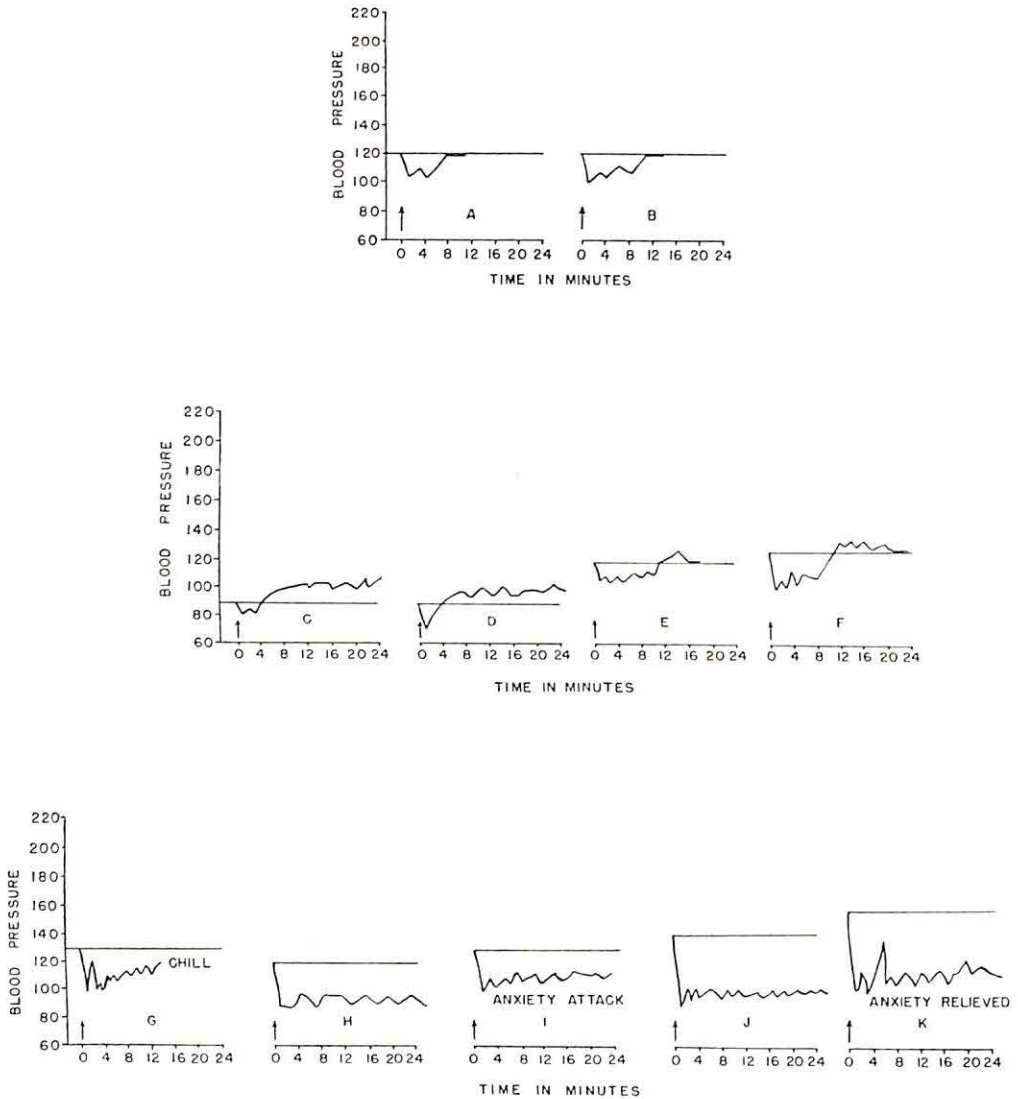


Fig. 1. The blood pressure reaction to intramuscular injection of 10 mg. of mechlorol in 11 different tests:

A and B. Normal controls. Homeostasis restored within 10 minutes.

C, D, E, F. Rebound with overshooting over preinjection level, such as found in aggressive psychopathic personality, obsessive-compulsive neurosis, and in certain cases of schizophrenia.

G, H, I, J, K. Prolonged fall of blood pressure with failure to reestablish homeostasis within 25 minutes, such as found particularly in manic depressive or involutional depressions, but also in schizo-affective psychoses and certain schizophrenics. (Modified from Funkenstein et al.¹²)

anxious clinically—"do not recognize any familiar symptoms of their anxiety when experimental stresses are placed upon the autonomic nervous system." The latter is the case particularly in schizophrenics.

In their observations they were able to establish significant relationships between autonomic pattern, subjective reaction and the patient's prognosis with various forms of physical treatment. Successful treatment was invariably associated with a changed autonomic pattern. They found that those patients in whom mecholyl evoked anxiety or a chill as well as those in whom mecholyl relaxed anxiety tended to improve following electroshock therapy, whereas those in whom epinephrine precipitated anxiety were made worse by electroshock,⁹ while at the same time the epinephrine-precipitable anxiety increased concomitant with the clinical aggravation of the patient's symptoms. It has now been established that many patients in whom epinephrine evokes anxiety respond favorably, however, to insulin coma or subcoma treatment or to nonconvulsive electric stimulation treatment, the choice of treatment depending on additional factors and their combination.

The normal reaction to the injection of mecholyl (10 mg. intramuscularly) is a brief drop in blood pressure lasting no longer than ten minutes (Fig. 1 A and B); this is the reaction encountered in the vast majority of normal controls not under stress. If this reaction is found in the psychotic patient he tends to remain unimproved following electroshock therapy. A slight or normal drop in blood pressure followed by an overshooting response may also be found among normals but is particularly frequent among patients with aggressive psychopathic personality (Fig. 1 C, D, E, F), in severe obsessive compulsives, and in certain schizophrenics. Patients responding to mecholyl with this reaction tend to remain unimproved by EST or any physical treatment. An immediate paradoxical rise of blood pressure on injection of mecholyl is very rare (see Chapters XIII and XX).

A reaction to mecholyl that involves a prolonged, moderate, or marked fall in blood pressure with failure to establish homeostasis within twenty minutes (Fig. 1 G, H, I, J, K) is most frequently found among patients suffering from depressions and involutional psychoses, but particularly also in schizo-affective psychoses and even among certain schizophrenics, particularly in recent and acute phases of the disease. Patients evincing this type of reaction—especially when it is accompanied either by increase or relaxation of anxiety or by a chill—tend to improve following EST, irrespective of psychiatric diagnosis.

The reaction to epinephrine injection (0.025 mg. intravenously*) allows an additional clinical grouping, which is, however, based more on the emotional and clinical reactions of the patient to the injections than it is on the blood pressure response itself, except in the case of insufficient response (Fig. 2 C), which does not occur very often in psychotic patients and then only in schizophrenics, particularly catatonics. Patients who show this type of inadequate reaction are unlikely to benefit from EST; in one case in my experience, however, such a patient responded well to insulin coma treatment. Normal subjects may show either moderate or marked blood pressure reaction to epinephrine (Fig. 2 A, B).

Patients in whom epinephrine elicits a moderate or marked blood pressure response combined with an anxiety attack (Fig. 2, D, E) tend to be made worse by electric shock. They do, however, respond well to nonconvulsive electric stimulation and to insulin coma or subcoma treatment.

* Funkenstein originally used 0.05 mg., but later recommended 0.025 mg., which is the dose that I am using exclusively.

It should be kept in mind that when we speak of enhanced reactivity to either mecholyl or epinephrine, what this enhanced reactivity actually indicates is an increase in the general tone of the opposing autonomic

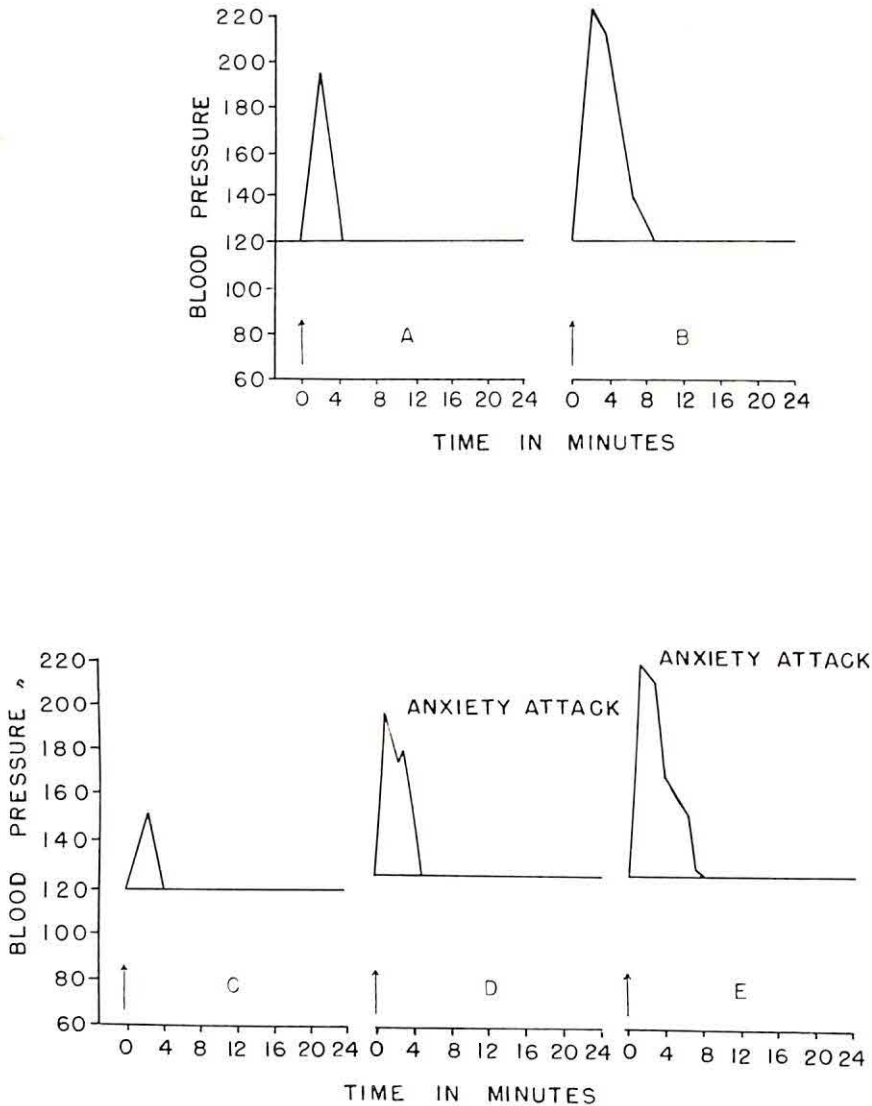


Fig. 2. The blood pressure reaction to intravenous injection of 0.05 mg. of epinephrine in 5 different tests:

A. Moderate rise without precipitation of anxiety; normal control.

B. Marked rise without precipitation of anxiety; normal control.

C. Minimal rise, such as occurs only in schizophrenics, without precipitation of anxiety.

D. Moderate rise with anxiety attack at height of reaction such as may occur in various types of psychoneurosis, manic depressive psychosis, involutional psychosis, and schizophrenia.

E. Marked rise with anxiety attack at height of reaction such as may occur in various types of psychoneurosis, manic depressive psychosis, involutional psychosis, and schizophrenia. (Modified from Funkenstein et al.¹²)

system. An enhanced reactivity to mecholyl indicates an increase in the general adrenergic tone, while an enhanced reaction to epinephrine may indicate an enhanced general parasympathetic tone.

This relationship is further complicated by the fact that the sympathetic system acts through two transmitting agents rather than only through one as believed formerly, one being epinephrine, the other norepinephrine. Most physiologists believe that norepinephrine is identical with Cannon's sympathin E. Excessive secretion of epinephrine is neutralized by mecholyl, whereas excessive secretion of norepinephrine is not neutralized by mecholyl.¹⁴

One of the most interesting findings of Funkenstein and his coworkers has been that when either the physiologic or the psychologic picture changes, the other changes simultaneously, the tie-up between psychologic state and physiologic state being as a mirror image. A change in autonomic pattern takes place following any form of treatment that has led to a change in the psychologic state or when such a change occurs spontaneously. Otherwise, the same autonomic pattern tends to persist in any individual as long as physiologic and psychologic conditions remain the same. If one changes alone or independently, relapse is usually inevitable.

It might also be noted that Funkenstein found the pattern of autonomic reactivity to show pronounced differences in psychotic and nonpsychotic phases among a small group of patients whose histories were marked by asthmatic attacks in nonpsychotic intervals and by freedom from asthma when psychotic, and in one case in which urticaria was associated with the development of a paranoid psychosis.¹⁶ Those patients who could be tested in both phases developed acute asthmatic attacks on injection of mecholyl and showed increased parasympathetic reactivity; when psychotic they reacted to mecholyl with mild, fleeting attacks and showed autonomic patterns which indicated improved parasympathetic homeostasis and sympathetic overactivity. (Mecholyl precipitates asthma only in persons with a history of asthma.) In the patient who showed an association between paranoid ideas and cholinergic urticaria, mecholyl provoked urticaria while he was psychotic, but had no such effect when he was nonpsychotic. It might be noted in this connection that McAllister and Hecker³⁷ found that a far smaller proportion of psychotics (schizophrenics, manic depressives, organics) showed physical signs of allergy during hay fever season than did the normal controls and the epileptics.

An additional yet unexplored avenue along which the work of Funkenstein and his coworkers may be leading, is that of finding out more about stress reactions among normals that might provide a basis of prediction of mental breakdown under certain conditions of stress, similar to the work of Ivy²⁸ in regard to peptic ulcer and that of Wolff^{50, 51} in regard to hypertension.

It thus appears that the physiologic accompaniments and manifestations of mental disorder may fall into three groups: Those which shift simultaneously with shifts in the psychologic picture and which thus may be interpreted as the physiologic aspect of that particular mental state; those changes resulting from a predisposition to instability that may be reflected in marked reactions to stress (physiologic or psychic), leading to intensified and prolonged abnormal reactions and possibly finally to altered, abnormal patterns; and those changes arising primarily from chronic emotional stress and rigid basic attitudes.

Such defective physiologic functioning—whether arising on a genetic basis, whether resulting from early conditioning, or whether the outcome of chronic emotional states—is increased by psychic or somatic stress and

may lead to changes both in the nervous system and in the general physical state. Those changes that take place in the nervous system may be expressed in faulty metabolism and defective enzyme patterns associated with lowered nerve excitability and decreased power to generate and propagate current along the nerve, in lowered intensity of metabolic activity, and possibly ultimately in abnormal neuronal discharge and faulty nerve functioning in general. In this connection one is reminded of Meadow's and Funkenstein's finding that among schizophrenics certain autonomic patterns are associated to a statistically significant degree with a tendency to think concretely rather than abstractly (Meadow and Funkenstein.^{38, 39} Those changes that take place in the physical state may be expressed in decreased responsiveness and adaptability to outside influences, in lowered energy level, and in inability to tolerate stress—obviously the effect of such changes on social relations will be far-reaching. In sum, then, all these changes contribute to greater physical and social maladaptation and to poor adaptive adjustments.

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IV

THE CULTURAL BACKGROUND OF MENTAL DISEASE AND OF ATTITUDES TOWARD ITS TREATMENT

THE INFLUENCE OF CULTURE ON MENTAL DISORDER

Broadly speaking, the culture into which a person is born defines his world of reality, its contents and its dimensions. It provides bases and focal points for perception, for determining what, in the terms of Gestalt psychology, will be focal point ("figure") and what background ("ground") (Murphy²²); how time and space will be perceived, how "reality" will be apprehended, the nature of relations with the self. A culture is made up of the prescribed, accepted, and approved ways of behaving, reacting, dealing with eternal human needs, urges, relations with others.

As cross-cultural studies have grown in scope and in scientific sophistication, the amazing plasticity of human nature in absorbing and performing multiple divergent patterns of behavior has become increasingly apparent, and the extreme importance of early training methods carried out by parents according to the cultural standards of child care is gaining wide recognition (consider the work of Kardiner,¹⁴ Erikson,¹⁰ Bateson and Mead,^{7, 8, 17, 18, 19, 20, 21} to mention only a few). Erikson,¹⁰ for instance, points out that the culturally prescribed ways of dealing with early organic modes of expression—which have their counterpart in complex social modalities—are an extremely important influence in how the individual approaches and deals with life; the experience the child has in these early phases throws weight on one side or the other in certain nuclear conflicts—such as, for instance, on the side of confidence and initiative in early attempts at locomotor and sphincter autonomy or on the side of self-doubt and mistrust.

At the same time these cultural studies make it plain that the malleability of human beings is not infinite, that certain patterns of reaction are common to all men, that no one cultural group has, for instance, unique defensive operations, but that all men are given to repression, projection, introjection, to psychosomatic reverberations, that all share certain basic biologic needs and certain preferred ways of dealing with them, although the exact content and form of reactions may vary exceedingly from one group to another.

Aside from the effects of child-rearing methods on basic character dynamics, it becomes apparent that in actuality the outer reality with which all people must deal is essentially a world patterned by social-cul-

tural precepts and interactions—or to phrase it in another way, when we refer to cultural-social precepts and interactions we describe the world we live in with others in our group. This, then, is an extremely important aspect of the “outer reality” with which the personality must deal. It is a third abstraction from a total process, the third member of the trichotomy, mind, body, and outer world.

The prescribed ways of the outer world, however, are not always mutually harmonious and in certain dynamic, heterogeneous cultures like our own may be numerous and conflicting. European-American civilization is heir to a tremendous complexity of subcultures arising from many and divergent sources, giving rise to many and conflicting standards. It is a dynamic, changing civilization full of extremes which often serve, however, to conceal broad similarities. It is a culture replete with conflicting ways of looking at and dealing with life and surfeited with frustrations of one kind or another. In some ways, our culture is best comprehended from a historical perspective, since history reflects many of the changes in mores and in modes of thought—although often not the most basic and implicit assumptions and axioms.

As Erikson¹⁰ has pointed out, however, the frustrations implicit in a cultural scheme are not necessarily disrupting provided they appear meaningful to those frustrated, and this fact carries special impact in the frustrations imposed in methods of rearing children. The sense of “identity” which is essential for a feeling of continuity in one’s life is imparted by the social meaningfulness of one’s role and by the basic and implicit assumption of worth-while purpose.

Since, moreover, an individual’s sense of approval from others as well as from himself depends in a large degree upon how well he is able to integrate conflicting standards in a socially acceptable way and yet fulfill his own needs, the degree of complexity of a given culture together with its ready-made compromises will have significant bearing on its potentialities for mental health or pathology.

In a sense, mental disorder is defined by the culture in which a person lives—that is, whether the individual’s reactions are recognized as abnormally deviant by the members of the group according to their standards of behavior and often whether they are so recognized by the affected individual himself. Certainly deviation as such is not the sole criterion in most societies. It is the meaning assigned to the deviation that is significant—that is, whether the deviant members are regarded as saints, geniuses, or lost demented souls; whether hallucinations are regarded as symptoms of grave pathology or as supernatural visitations.

Similarly in a given society the same disorder may be differently regarded at different times. A good example might be the role of a borderline paranoid in our society. Often such paranoids appear to have an intense, even exaggerated sense of purpose which may be extremely useful in some phases of social development but quite disruptive in others. They may serve the valuable function of innovators and crusaders for social justice or may simply be eccentric and queer, depending on the social need in the cause they espouse as well as in the more individual aspects of their behavior.

On the other hand, there is no doubt that mental disorder does exist

quite independently of whether or not the condition is socially recognized as such.

The influence of a culture on the production or intensification of mental disease has not, so far as I know, been studied systematically or quantitatively in such a way as to lead to significant statistical analysis. Nevertheless, on the basis of observations, impressions and, more importantly, our knowledge of the role of stress, conflict, and anxiety in mental disorder on the one hand and the significance of personality strength and resources on the other, it is possible to make certain assumptions about this matter.

From the viewpoint of relative balances, it becomes important to consider the amount and quality of stress the members of a society are subjected to as balanced by the satisfactions they may obtain; the amount and kind of anxiety-provoking conflict they may be exposed to without the provision of satisfactory solutions or compromises as balanced against such factors as: the amount and quality of basic security provided in childhood and familial ties; the nature and depth of affective ties with other people that the societal set-up fosters; the prevalence of various measures that promote healthy personality development and integration; the general level of emotional security in the society (and hence in the parents)—which in turn may be based to an important degree on the stability of the mores; the implicit sense of purpose in the prescribed ways of life; and the amount of basic gratification and satisfaction offered to both fundamental biologic needs and to more highly evolved social needs.

Similarly, the influence of a culture on mental disorder may have qualitative aspects, chiefly, probably, in the specific content of conflict and symptoms and in the types of defensive operation most readily accepted by other members of the group. Horney has been especially concerned with this qualitative, specific content and the reaction patterns prevalent in our present-day American society. Theoretically, the different character structures arising from various methods of child rearing and from differing cultural values in different groups may lead to somewhat different dynamic processes or balances—although not too much is actually known about this, while on the other hand the universality of certain personality reactions and defensive operations is quite well established, even though the nature of the conflict or stress may differ strikingly in different groups.

Suicide, for example, follows strikingly different patterns in different cultural groups.^{5, 11, 16} In some parts of Europe and China, as well as in Japan, outstanding loss of face or prestige makes suicide socially acceptable, if not mandatory, particularly in the young adult and middle-age groups, in order to save the family from disgrace. If such a man went to prison, his family would be social outcasts. By his suicide, however, he frees them from such a fate both because his guilt cannot then be proved and because he will have atoned for any misdeed and expiated his guilt according to the standards of his society. This undoubtedly is related to the more closely woven unit of the clan in European, Chinese, and Japanese society, but there are certain other implications of the philosophy with which life is faced. In America or England, for instance, such suicides are regarded as disgraceful and cowardly, leaving the family to bear the brunt of the problem to come.

In our society suicide is more acceptable in certain other groups such as

the aged and incurably ill. Their loss of power, status, and the capacity for enjoyment and satisfaction, if not actual suffering, are regarded as understandable and acceptable motivation for self-destruction. On the other hand, suicides among the aged have hardly been heard of in China, Japan, or Europe, where the tightly knit clan structure supported the old man and invested him with status.

From the point of view of individual motivation it would be interesting to investigate in different cultural groups the nature of the introjected image that the neurotic suicide hates so much that he kills himself to kill it.

Abrupt shifts in societal values—as in times of great emergency, war, and carnival—may have dramatic effects on certain neuroses which for all intents and purposes are then cured. The sanctioning or approval of hostility in time of war, for instance, has a remarkably alleviating effect on some anxiety neuroses based on repression of hostility. A patient who lived in a perpetual state of suppressed rage and frustration to which she reacted with anxiety, shame, and depression showed distinctive improvement when the Korean War started. She consciously experienced the onset of war as a release and realized its connection with her considerable lift in spirits (this patient has had four years of psychotherapy as well as various forms of physical treatment, all of which failed to bring about any sustained remission). Similar reactions were observed in England when severely disorganized neurotics became excellent air-raid wardens. Apparently the implied approval of hostility and its easy, socially approved displacement removed the need for neurotic defenses against their hostile aggressiveness, and they were able to function efficiently and to endure hardship and stress.

In the social atmosphere of the Nazi regime the same sort of "cure" took place in certain neurotics who found scope and support for their destructive drives that hitherto had been vigorously defended against. In the case of one scholarly, rather passive man, a physician who suffered from peptic ulcer and a domineering wife, the Nazi philosophy relieved him of all self-doubts and convinced him that he was indeed a superior racial being. He was able to throw off his passivity, his ulcer, and his wife and take up new patterns of aggressive masculinity and outright sadism which he easily justified Nazi-wise. After his capture by our Army and during his trial in Nuremberg, first his meekness, then his wife, and shortly thereafter his gastric symptoms returned. I am sure that in the setting of militant communism in Russia where strong aggression is directed against the outside world such shifts can also take place. However, while they have seemingly curative effects in certain individual types of neuroses, it goes without saying that such shifts in social attitudes are by no means conducive to greater mental health in general.

What has actually happened in such situations is that the whole group has become subject to group psychosis. It is important for us to realize that such group psychotic developments actually exist. We are entitled to call such social states of mind as the Nazi one, group psychosis because of the parallel between the dynamic psychologic changes that occur in the individual psychotic and in such social groups—there is a marked lowering of defenses against sheer, primitive destructiveness; the elements of control

and integration vanish; reality testing is corrupted, and reality sense distorted; a rational, constructive approach is decried and glaring contradictions may dwell side by side in amity (e.g., asceticism is vaunted at the same time the most primitive instinctual indulgences are encouraged; personal responsibility for actions is denied). In such a setting, neurotic defenses and symptoms are drowned in the upsurging of primitive instinctual forces, just as the psychotic patient loses his neurotic characteristics in the far more primitive, disorganized personality shifts that have now taken place. (The gradual development of these shifts has been pointed out elsewhere by the author.^{1, 2, 3})

SOCIAL ATTITUDES TOWARD MENTAL DISORDER

The other side of the coin, from the cultural point of view, is concerned with how mental disorders are regarded and treated, and this seems to depend on certain group aims and assumptions and the attitudes that develop out of them. Cutting short any cross-cultural comparisons, it is pertinent to consider our own cultural biases, which mostly have become apparent in retrospect, for we are seldom fully aware of some of our most basic assumptions while we are still functioning under their influence. It is not until they begin to outwear their usefulness and no longer square with our gradually developing picture of reality that we are inclined to trace back and discover them as sources of error.

While our basic scientific philosophy and approach (which will be discussed in Chapters VI and VII) are perhaps among the most important of our cultural biases regarding how to treat mental disturbances, it may be worth while to mention a few of the basic assumptions and premises that are part of our cultural fabric. Perhaps most significant among these, from the point of view of psychiatric treatment, is a humanistic orientation based on implications of the importance, worth, and dignity of the individual, regardless of his mental or physical state, and, associated with this, the responsibility of society for the care of those members who cannot care for themselves, with the assumption that every individual is entitled to at least a modicum of happiness and security. Following closely upon this is an assumption now receiving increasing attention and study—namely, that treatment proceeds through and requires human contact, a recognition that social relations are basic to human life.

Tied to our conception of mental abnormality is an implicit assumption of a norm of ideal adjustment and integration—which seems to be something in the nature of a Platonic ideal. Though seldom found in clinical experience, the ideal of normality of function is an important tenet in treatment as well as in preventive measures and child rearing. Related to this is our obvious conviction—facetious as well as fanatical comments to the contrary notwithstanding—that sanity is to be preferred to insanity, reality to unreality, healthful function to dysfunction. Strange as it may seem, a paradoxical view has been taken by some extremists of the psychodynamic school that recovery should not be forced upon the psychotic by physical means such as shock therapy or leukotomy when recovery would run counter to his dynamic needs (Atkin,⁶ Zilboorg²⁵), although these needs are certainly merely a part, probably even the most important part, of his disease.

By this logic we would not be allowed to relieve an unconscious patient from an acute traumatic brain compression, since any activity runs counter to the dynamics of the unconscious state. In order to avoid confusion, we must keep firmly in mind that ethically, morally, and legally all reality-oriented decisions as to the treatment of an unconscious patient as well as of a patient suffering from mental disease rest solely with the next of kin or legal guardian of the patient.

We also assume a certain uniformity of reactions among people; this is the basis of classification and study of trends in groups. While this assumption has been essentially verified in experience, yet it often leads to certain erroneous tendencies in forming logical conclusions in diagnosis, etiology, and type of treatment. As Cameron⁹ points out, recent psychosomatic studies, particularly those of Selye,²³ and Wolff and Wolf,²⁴ indicate that the reaction repertoire of the human organism is relatively limited: "The organism is able to react in only a limited number of ways to noxious stimulation, and . . . many different kinds of noxious stimulation may produce the same effects in the organism." In other words, similar conditions may rise from different beginnings; conversely, similar factors may give rise to dissimilar effects in different individuals.

Another important consideration is our belief in modifiability—physical, emotional, intellectual, social—although various aspects are seen as more modifiable at one period of research than at another. Cultural factors and socio-political motivations are strong determinants of our basic attitude toward the modifiability or unmodifiability of mental disorders.⁴ The rigid atmosphere of German determinism, for instance, favored an academic belief in the unmodifiability of abnormal traits. American social trends with their numerous evangelical roots favor a strong belief in a high degree of modifiability, including change by mental and spiritual means. The Russian system also favors a belief in the possibility of modification by social forces and social compulsives.

There is no doubt, for example, that the Russian government is playing down genetics because a firm belief in the modifiability of human beings is required as a source of morale in a society of its type. On the other hand, we too play down genetics and probably for the same reason: a free, developing democratic society also needs a firm faith in the free unlimited potentialities of its members—that is, a similar belief in modifiability. In Nazi Germany the situation was reversed: their morale was based on a compensatory belief in innate superiority that necessarily presupposed unmodifiability; accordingly the German government emphasized the findings of genetics with their deterministic implications.

Nevertheless, by no means all actions are determined by the professed beliefs and expressed preferences of prevailing scientific theory. While the Nazi government sponsored and emphasized genetics, thus stressing unmodifiability, including psychological unmodifiability, making it a rationale for its racial policies, yet at the same time in dealing with its own adherents the party acted as if it were thoroughly convinced of the highest degree of modifiability—psychologic and otherwise—as may be witnessed by its techniques of propaganda, training, and indoctrination.

Similarly, there are alternative and opposing trends to our basic belief in modifiability, explicitly and implicitly expressed in our own social code

and in certain legal traditions that presuppose a concurrent belief in unmodifiability. Thus conflicting social trends may live side by side.

The attitude of scientists—objectivity notwithstanding—is of course strongly influenced by these major and minor social trends and resulting conflicts. In a free democratic society, in spite of the ebb and flow of the major trends in social attitudes and resulting trends in scientific attitude, a sufficient reserve of opposing trends remains viable and thus may be taken over and reexamined when the direction of thought again changes.

In our own society at present, our success with the new techniques of psychologic understanding and treatment—in spite of the fact that many of them require nonpsychologic measures as adjuvants—has produced in our scientific circles a trend to overrate and overstate our ability to modify mental illness by psychologic means. This trend goes on in spite of the fact that even now no scientific proof has been established that the major mental illnesses are at all significantly modifiable by purely psychologic means. This is an example of how major trends of scientific endeavor go on for a time essentially unchecked by the impact of reality.

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One of the most important biases both in how we have regarded and how we have treated mental disorders has lain in a type of scientific approach used in the hope of gaining knowledge by abstracting and isolating aspects of an ongoing process, holding them static and out of context by imposing "scientific controls," and on this basis attempting to determine what is cause and what effect. The aspects thus abstracted have tended to become concretized as discrete realities, a tendency that has resulted in endless and sometimes meaningless questions and problems.

Among the most important of these concretized abstractions in psychiatry have been many of our diagnostic categories, as well as those now untenable divisions: mind-body, heredity-environment, normal-abnormal, structure-function, mechanism-vitalism, neurosis-psychosis. These divisions represent an artificial abstracting and isolating from one another of reciprocal relationships that in life processes cannot be divorced. Such artificial isolation of one factor from an organic whole or of extremes from a continuum is scientifically justified and valuable when it leads to further understanding of and research on the whole process, but the artificiality of the abstraction, of the isolation of the function or process, must always be borne in mind if we are to avoid the fallacy of misplaced concreteness, of reifying an abstraction of our own minds.

Much psychiatric diagnosis still is based on rather superficial classification derived from the isolation of certain features that seem to occur with statistical regularity in mental disorders and the grouping together as disease categories of certain patterns that seem to emerge. These neat medical classifications have never proved entirely satisfactory, however.

As the deficiencies of this scientific approach with its almost inevitable concretizations became increasingly obvious, another approach appeared that at this stage in our scientific development seems much more meaningful and fruitful. This is called by some the field approach, and implies consideration of dynamic reciprocal processes of influence and counter-influence within a field of force (after the model of physics), so that not only the individual contained within a skin is considered, but also all the influences impinging on him and that he himself impinges on. Event sequences rather than specific causative beginnings are considered (Cameron¹). The older methodologic approach and reifications still hang on, however, in spite of our good intentions and still color much of our thinking.

Take for example, the prevailing dichotomy between neurosis and

psychosis, in many ways a valuable division. Both are mental disorders—that is, they involve some deviation of mental functioning from the ideal in integration, energy, symbolization, emotional balance, control, reality testing, and so on. Their essential similarity was recognized by some of the early modern psychiatrists who did not try to pigeonhole them into neat medical classifications but regarded them broadly as disturbances of the soul, alienations of the mind.

Gradually, however, the differentiation now expressed in the two words emerged, at first in terms of necessity for custodial care and in medicolegal considerations, later in terms of their meaningfulness or comprehensibility, the indications for treatment (especially in terms of accessibility to therapy), and on the basis of their presumed cause—unknown physical ones in psychosis and “imagination” in neurotic disturbances (McDougall³).

Aside from the practical usefulness of this division, however, it must be admitted that the distinction between neurosis and psychosis is not a clear-cut one, not only in terms of the strange borderland between them, but in the gradual shading of one into the other in a sort of continuum of severity of pathology (as expressed in symptomatology, disturbance of function, contact with reality), and in the progressive transition from one state to another in the same individual over a period of time, especially if the first evidences of disturbance become disabling in adolescence or early adulthood.

While most of the criteria that have been established to differentiate neurosis from psychosis are useful as practical rules of thumb, none is invariable or even distinctive in the majority of cases (although their combination may be). One of these, for instance, has been the extent of the patient's insight into his illness, which is thought to be more often present in neurotics. The opposite, however, is remarkably often true. There are many psychotics with keen insight and many neurotics quite devoid of it. The same may be true of contact with reality, for the reality of the neurotic is often a rather hazy, personalized one, and he frequently merely gives lip service to it.

Today, therefore, this differentiation is again beginning to dissolve, particularly in recognition of the psychologically meaningful aspects of psychosis. Neurosis and psychosis are coming to be viewed, rather, as a system of receding lines of defense and fortification, graduating from the neurotic reaction as the first line of defense, to the severe mood disorders as the next line of defense, and finally to the content disorders, the schizophrenias, as the last line of defense; and the different diagnostic categories in neurosis are seen not as discrete entities but as various balances and emphases in type of defensive operation.

From this point of view the difference between neurosis and psychosis is not so much qualitative as quantitative, in that individual cases in the two categories may show varying degrees of personality disorganization and malfunctioning, distinguished from each other in such quantitative terms as: degree of social and emotional impairment of relations with others, degree of distortion or loss of sense of reality and ability to test reality, responsiveness to therapy (and hence hope in prognosis), type of defensive operations resorted to—in what balance and intensity and the consequent degree of personality crippling.

Supporting such a view may be certain aspects of the individual course of mental disorders. Certain early neurotic states are found, on follow-up studies, to have developed into psychosis, and certain less severe psychotic or partially psychotic states often subsequently deteriorate into more severe disorganization. The reverse process may also occur. A schizophrenic patient who has responded favorably to insulin coma treatment may in a subsequent period of stress develop a pure depression or even a pure anxiety neurosis, reflected not only in the clinical picture, but in the accompanying reaction patterns of the autonomic nervous system.

Rennie⁶ found when he investigated twenty-five year follow-ups on cases originally diagnosed as psychoneurotic that, contrary to general belief, a good many had developed psychotic episodes with the passing of time. Similar follow-ups on Hoch's² cases of benign stupor (Rachlin^{4, 5}) that occurred in the teen age and twenty-year age group showed that most of these had deteriorated into schizophrenic reactions. At the same time we know that certain depressions, especially in people in their twenties, later develop into a schizophrenic illness. It is a telling reflection on the period of rather formal and superficial classifications, that the majority of Kraepelin's manic-depressives in the younger age groups were found, on follow-up studies, to have developed schizophrenia. It has also become common psychiatric experience that certain neuroses in the teen-age group, especially those with obsessive and phobic manifestations, eventually decline into schizophrenic psychoses, as do most of the depressions and manic episodes of the teens and twenties. Then, too, consider the still mysterious borderland of the psychosomatic patients, most of whom clinically appear clearly neurotic, and whose relation to psychotics is extremely provocative.

To be sure, for practical purposes we cannot dispense with our diagnoses entirely, but we must be aware of the fact that even from the cross-sectional viewpoint they express only one element of the problem, while longitudinally they represent shifting strategic positions in the patient's defensive battle between his anxiety and his conflicting drives.

Diagnosis of the psychiatric category alone is not enough even for a cross-sectional diagnosis made in order to determine the therapeutic line of action. We must also take into account the physical functioning, the psychosomatic reverberations—whether or not such reverberations are mediated by the adrenergic or the cholinergic system—the social setting, the patient's intelligence, and his personality assets. When all these factors are considered, the practical diagnosis that emerges indicates both where the patient stands in this battle against his disease and what sort of help he needs.

Depending merely on the psychiatric diagnosis may be dangerous or misleading for several reasons—one is the tendency to isolate and reify the process from its context in life; another lies in certain assumptions behind various diagnoses, the most important of which may be the tendency to conclude that similar states arise from similar causes and that similar syndromes will respond to the same type of treatment—another case of isolating process from context; a third, related difficulty lies in the diagnostic classifications themselves and their frequent basis in aspects of the disordered process that are not necessarily relevant to the treatable aspects. Even though important facets of the picture may still be

missing, if we consider all the data involved in order to assay their possible relevance. we are more likely to get an accurate, rounded picture of the processes involved.

Accordingly, it must be admitted that sometimes the purely psychiatric diagnosis, though accurate, may be the least helpful one in determining the potentialities of the dynamic situation in which the patient finds himself, as will become more evident in subsequent chapters. In certain cases the state of the autonomic nervous system may more accurately represent the main issue, or at least that point at which we can most constructively interfere with the dynamic sequence. I have, for instance, treated manic patients by three different methods, all with equal success. the choice of treatment depending on factors in the picture other than the psychiatric diagnosis alone. I have found that manic patients whose tension and abnormal drive are associated mainly with excessive responsiveness to adrenergic stimuli respond favorably to nonconvulsive electric treatment; those whose chief autonomic abnormality lies in their excessive reaction to cholinergic stimuli respond to electroshock treatment; while those manic patients whose autonomic nervous systems are functioning reasonably normally are best treated with psychotherapy alone. In the days before Funkenstein's system of autonomic diagnosis was available, I occasionally made a manic patient worse by treating him with electroshock, as well as occasional patients with depression.

Diagnosis in the sense of understanding processes is still the main basis for determining type of therapy, but such diagnosis must be multifactorial. rather than being a single label based solely on clinical syndromes, which no matter how accurate cannot take into consideration all the factors in the actual situation.

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Like all medical procedures, psychotherapy contains elements of art as well as science. Actions and attitudes are frequently more important than words in the practice of psychotherapy.

Emotionally sick people have much in common with children, particularly in that their complaints and actions often express their feelings and problems in a symbolic manner, frequently they distrust words, they learn more from example and by identifying with good models than they do from precept, and they tend to relate to the world more according to emotional experience than by instruction. Thus often more important than *what* the therapist tells the patient is *how* he acts toward him, how he listens, and how he speaks. Frequently it is more valuable to offer a new and good interpersonal experience than to offer new thought content, although both are inextricably interwoven.

NONVERBAL PSYCHOTHERAPY

Quite apart from its verbal aspect, psychotherapy involves a primary, nonverbal element (Alexander³). In fact, in its most elemental nonverbal form, psychotherapy can even be carried out successfully on animals. Translated into terms of the animal trainer, Pavlov¹³ (see also Konorski¹⁰) in his work on experimental neuroses found essentially that in treatment it was necessary to build up dogs with "weak" nervous systems (a term by which he designated those animals who reacted to stress with inhibition, a sort of passive folding up); while those with "strong but unbalanced nervous systems" (those animals who reacted to stress with excitation, a pronounced readiness for action whether fight or flight) first had to be broken or "reduced," by sedation for instance, and then built up. Obviously, in treating animals, attitudes and actions play the main part, since animals do not understand words.

It is this ability to influence animals or human beings by attitudes and actions that is the most important natural asset of the psychotherapist. If I were asked to devise a screening test to discover those gifted for psychotherapeutic work, I would say that the sort of person most likely to become a good psychotherapist is the one whom stray dogs take to, who can gain the confidence of a beaten dog, or who could easily ride an anxious horse, sensitive to the bit, that customarily shied at the slightest provocation. I would place far more emphasis on these faculties and attitudes—which

are inherent as well as acquired—than I would upon mere knowledge of psychodynamic theory.

Effective Actions and Attitudes

The actions and attitudes that have been found most beneficial on the basis of psychodynamic understanding are remarkably similar to, often identical with, those derived from true religious attitudes. Modern psychiatry has rediscovered the importance of love, in the spiritual sense of the word, as a therapeutic force. As Alexander and French¹ point out, the priest in Victor Hugo's novel *Les Misérables* performed a masterpiece of brief psychotherapy when he brought about the reform of the hero, Jean Valjean, by his attitude and acts of unexpected kindness after Valjean tried to rob him. It is of fundamental importance that age-old spiritual insight and modern psychodynamic insight have led to the same conclusion: that certain thieves are deprived people who must first be given to, in a sense treated with loving-kindness, before they can be reformed. The fundamental underlying attitude in good psychotherapy is a basic kindness, concern, and good will toward others, expressed with warmth and force, and applied with enthusiasm. This attitude is most effective if supported by the skillful use of essentially nonverbal, primary means of interpersonal communication (Alexander³). Eissler⁸ has recently reemphasized the therapeutic efficacy of these elementary approaches—e.g., “the archaic language of gesture and purely vocal quality”—which transcend in importance the cumbersome system of secondary processes with their rational, logical, verbal communications. These nonverbalized means of effective interpersonal communication are of course independent of the doctrinal and conceptual aspects of the psychotherapeutic schools or methods otherwise adhered to by the therapist. This should be kept in mind in evaluating the results of any therapy.

It is therefore important to be aware of these nonverbal and primarily unformalized attitudes. With some people they are a natural gift, but they may be developed through an awareness of their importance and a scientific study of the way they facilitate successful work. The psychotherapist must keep constantly in mind that his actions and attitudes are at least as important as his words.

TECHNIQUES OF PSYCHOTHERAPY

Once this fundamental principle has been understood, the various techniques of psychotherapy may be learned, but they are of little avail without a basic attitude of concern and kindness. Essentially these techniques are: (1) supportive treatment; (2) ventilation; (3) abreaction; (4) shift of emphasis; (5) interpretation. Specialized techniques, whose elements are inherent in all psychotherapy, including the five aspects just mentioned, are an additional four; as separate procedures these are usually limited to certain specific situations and conditions. They are: (6) suggestion; (7) persuasion; (8) hypnosis; and (9) reassurance. It is of particular importance, of course, to know the limits of these various forms of psychotherapy and to know when to add other methods, such as medicinal treatment or the various forms of shock therapy.

1. Supportive Treatment. This type of treatment is especially valuable

in acute situational anxiety reactions, whether they are expressed in a fear of injury or in a fear of heart disease or of any other physical ailment. Here the main element lies in the attitude of the therapist. Regardless of whether the patient comes with an actual physical ailment or with a neuropsychiatric disorder manifested in physical complaints, it is of fundamental importance that the doctor listen attentively to the complaint, take it seriously, and subject the patient to a thorough physical examination. The thoroughness of an examination is frequently the main element in successful supportive treatment.

The effect of this type of treatment is, of course, enhanced by the physician's reputation in the community, by his standing as a professional man and citizen. A doctor's reputation and his thoroughness enhance each other; conversely, it can sometimes be disastrous if a well qualified and highly respected man takes the complaint of a patient lightly and because of his initial impression and a shortage of time subjects him merely to a hurried and superficial examination. Such treatment may increase the feeling of rejection that may be the main factor in the neurosis of an unsuccessful man or a rejected, unloved woman or child.

By his attitude and actions, the therapist must give the patient assurance that he cares for him, is interested in him, and wants to help him. There is an old saying to the effect that "a specialist is a man who does a rectal examination." I should like to paraphrase this in less graphic terms and say that a psychotherapist is a physician who takes a complete and thorough history, makes a complete and thorough examination, and plans his therapeutic program accordingly.

The verdict of physical health will be accepted by such patients only if they have been subjected to an examination that impresses them as sufficiently thorough, and in such examinations the physician must show by his attitude that he has an open mind and his concern is with finding the truth.

An important part of psychotherapy is teaching, and the most effective way of teaching adults as well as children is by example. If you want the patient to have an open mind and to accept the truth about his condition, physical and emotional, you must first show him that you yourself have an open mind and are interested in discovering the truth. If you have a good relationship with your patient, he will unconsciously imitate your attitude in this. The arousal of mimesis is indeed one of the most important instruments of successful teaching as well as of successful psychotherapy, and is particularly useful to the supportive treatment.

I do not, of course, mean to say that such supportive attitudes can overcome a severe, deeply ingrained anxiety neurosis or a prepsychotic panic, but an attitude of open-mindedness and thoroughness will be helpful even in those conditions, and it is always wise to withhold reassurance and interpretation until the patient is ready to accept them. Again, it is important to know one's limits. Unless you are very sure the patient is ready to accept reassurance and interpretation, both of which are double-edged swords to be used with great caution, it is much better to help the patient stumble on the solution by an attitude of thoroughness, open-mindedness, and interest in the true state of his health—in essence, a reinstating of the necessity for reality testing. It is essential to realize that

reassurance is not always real support. The anxious patient who deep inside believes he is sick, dangerously sick, will often regard early reassurance as a brush-off on the part of the physician.

As I pointed out before, emotionally sick people, like children, have an uncanny sense of truth. The ability of children to look through and size up their parents and others close to them is frequently equal to that of trained psychologists and psychiatrists, and certain neurotic adults seem to retain this sensitiveness. True supportive therapy can never be given without the solid foundation of the patient's conviction that what the therapist says is truth. In supportive treatment the psychiatrist must decisively demonstrate that he is on the side of the patient in his battle with the world.

While support is, in varying degree, an element of all psychotherapy, it may become the main focus in certain conditions. In certain cases the primary therapeutic effort must be concerned with supporting the patient's present defenses and with enhancing his ego strength because of unmodifiable stress-producing external circumstances, including chronic incurable illness, or because the ego organization is already so weak that what is most needed is further support and strengthening of its defenses. The personality assets and strengths must be pointed up and attempts must be made to tone down, or reconstruct balances in, existing conflicts, without, however, doing this by analytic or insight-promoting techniques. Frequently it is exceedingly valuable to support and reinforce the reality sense, which not only may give a different perspective to the patient, but may increase his confidence and thus further fortify his personality.

2. Ventilation. It is of cardinal importance to give the patient full opportunity in a setting of understanding approval to ventilate freely his stresses and emotions in current and past situations. Once the patient is underway in effective ventilation, the therapist should interrupt as little as possible, merely standing by with the understanding and encouraging smile of a wise Buddha.

This technique is particularly helpful in certain cases of acute anxiety, anxiety hysteria, conversion hysteria, and in the anxiety states of the older age groups, especially those with hypertension, in which there is a good deal of suppressed rage—psychodynamically, whenever stifled emotions are, or should be, pressing for the release and outlet that the ego dared not permit in the original situation of danger.

Ventilation differs from abreaction in that the therapist plays an entirely passive and purely permissive role. It consists of giving the patient the opportunity to sound off, to explore relationships for himself, with a minimum of active participation by the doctor.

This technique, for instance, was quite effective in the case of a young woman who had suffered persistent throbbing pain in her cheek for three years, about which she consulted numerous doctors, dentists, and an otolaryngologist (who referred her to me), none of whom could find any physical basis for her complaint. Early in her interviews with me she remarked that this pain might possibly have something to do with "something terrible" she had done. From then on, through sixteen subsequent interviews, she poured out the grim story of her life, with very little interruption on my part except for occasional questions to keep the thread of her story in meaningful channels and to indicate understanding en-

couragement of her own struggle for insight. I refrained from offering any interpretation, but instead merely helped the patient by occasional questioning to find the meaning of her symptoms for herself. Her pain disappeared after the sixth interview, and later briefly recurred on two meaningful periods after the twelfth session. It again disappeared after the thirteenth interview and has not recurred in the subsequent five years.

3. Abreaction. It can frequently be observed that when ventilation proceeds on a purely intellectual level without expression of associated emotions, the therapeutic benefit is minimal or absent. After telling their story such patients may say, "Well, that's my story, but what's the use of rehashing it. I still feel the same." It is then that the therapist must induce genuine emotional participation, an emotional reliving of the stress situation for which the word "abreaction" has come into vogue, meaning literally "reacting off" or blowing off steam. The important thing is to remove the inhibition of thinking and feeling and allow the emotional experience to be relived. This will occur when the patient's defenses against the expression of these suppressed or repressed emotions are sufficiently lowered—as when he perceives the present situation as safe, quite removed from the dangers of the traumatic episode, or when his cortical inhibitions have been lowered.

There are various ways to bring about this abreaction. One is to go over the ground again, and by one's attitude, without direct urging, prompt the patient to give true vent to his emotions. The most important part of the attitude is to imply approval of a marked show of emotion. Sometimes the attitude is to imply approval of a marked show of emotion. Sometimes the use of drugs, such as barbiturates, ether, or benzedrine sulfate and its derivatives, or of special procedures such as hypnosis, have been useful in bringing about this needed reliving and working out of the emotional aspect of a traumatic experience.

Benzedrine, dexedrine, and desoxyephedrine, taken either orally or intravenously, appear to be more effective in the civilian setting than the barbiturates. On the other hand, the barbiturates have been particularly helpful in dealing with recent, severe stress such as that occurring during war or following other crushing-threatening events (Sargant and Slater¹⁵). Among such patients, abreactive methods have been most rewarding in those who reacted to such experiences with a state of profound inhibition. This inhibition can be successfully broken by the administration of the drug.

There is, of course, nothing particularly new about pharmacologically induced abreaction. Alcohol frequently accomplishes this result, and many sick people abreact themselves quite inadvertently by alcohol. Similarly, the principle of emotional abreaction has been used successfully, although probably unconsciously, by various ecstatic cults and sects. Mr. Bunn's snake cult in North Carolina is a recent and striking example in which touching a snake during the ritual produces intense emotional excitement associated with abreaction (Sargant¹⁴). The effect, however, can be produced more readily and more controllably by intravenous barbiturates, ether, or whatever.

It is, of course, important to realize that barbiturates, ether, or whatever the drug used, do not act in any specific way except in that they release cortical inhibitions. The drug in itself has nothing to do with the emotional response, which is in part provoked by the strong and still threatening stresses of the life situation as perceived by the patient (i.e., whether

"real" or subjective)—for instance, the imminence of return to combat—as well as by the pressure of strong but repressed emotional reactions to past trauma.

It is emotional charge that determines the intensity of the abreaction. The release of cortical inhibition by a drug is merely one of the trigger mechanisms to set off the explosive charge that must be detonated in order to free the personality. All of us who carried out abreaction treatments during the war and who later attempted to repeat them in a peacetime setting have been impressed with the marked differences between the rich emotional responses obtained in the combat setting overseas and the scant responses obtainable now, in veterans, for instance, more than six years after the traumatic episodes. The use of this particular method has consequently been limited in treating peacetime neuroses, in which in general the threatening situations are far less overwhelming and acute.

The greater the emotional charge attached to the traumatic material, the more readily emotional participation is bound to come forth when it is ventilated. It is probably for that reason that when traumatic sexual material is worked through with a patient it results in particularly dramatic abreaction and relief because of the intense emotional charge commonly attached to sexual matters. The same is true for long-suppressed rivalries and hatred, especially within the family circle.

Additional interpretation or synthesizing on the part of the therapist in working out the experience, while often beneficial, is sometimes of entirely secondary importance. I have seen severe inhibitory neurotic reactions significantly relieved by narco-abreaction even before the "synthesizing" treatment could be started, merely by the release of the tremendous emotional experience alone.

It appears that inducing the excitation, quite apart from the interpretation and working through of the emotion-laden material, has in itself a wholesome effect, regardless of how the excitation is produced, whether by the reliving of a recent crushing-threatening experience, by verbalization of old resentments and hostilities, by reliving sexual conflict, by participating in exciting ritual, or by touching off extreme excitation of the entire nervous system by an electric stimulus.

The physiologic aspects of abreaction as a form of excitation can, I feel, be best understood in Pavlovian terms. To be sure, Pavlov oversimplified; yet he also gave psychotherapy a physiologic correlate, a point of reference, a system along which relationships between psychotherapy and neurophysiology could be developed. From the Pavlovian point of view, one may regard this abreactive experience as a strong emotional stimulus that irradiates over the entire nervous system and frees the cortex and associated integrative functions from their inhibitory state. It is conceivable that the intense excitation of neurons associated with electric shock may be the extreme physical variant of the same principle, although in convulsive electroshock treatment the subsequent state of inhibition may be the more decisive factor—in which case, electroshock treatment should be considered a "reducing" process (in Chapters XII and XV there will be further discussion of the psychophysiologic aspects and psychologic meaning of EST).

In spite of the strong physiologic components of the experience, however, the emotional reliving and working through, the psychotherapeutic

guidance and understanding are extremely important. Despite all the lip service given to the unity of mind and body, many physicians still find it difficult to accept the fact that there is no separation of the psychologic and physical aspects of treatment; both should be fully utilized in a reciprocal interacting way.

We come now to the more specifically insight-promoting techniques having to do with interpretation, direct or indirect. Indirect interpretation is often preferable, particularly for the beginner and in the initial phases of treatment. It is very important not to overwhelm the patient with interpretation and thereby reinforce his defenses and resistances. He should first be cautiously felt out. It must be remembered that the scaling of strong defenses is a risky task in psychotherapy as well as in war. The wise general and the wise psychotherapist prefer a flanking movement; in the long run it saves time and risk.

4. Shift of Emphasis. The flanking movement that should precede direct interpretation and that often is sufficient to take the place of direct interpretation is shift of emphasis. By this I mean the subtle shifting of emphasis from the complaint or apparent conflict to the more psychologically significant material that the patient may have presented quite inadvertently without being at all aware of its import in relation to his complaint. It is wise to take as a starting point something the patient himself has related, focussing on an aspect that has hitherto been in the background of the patient's thinking.

Implicit in this method are elements of clarification and of restructuring the perceptual field. Two of its advantages are that it does not arouse antagonism and resistance and it permits the therapist to feel out the patient, to gauge his reactions. It is often particularly useful in current stress situations in focussing on a conflict which though conscious is considered by the patient to be unworthy of him, selfish, immoral, or disapproved by others, but something which in the permissive therapeutic setting can be recognized and dealt with without loss of self-respect. A frequent example is in marital problems.

Conversely, the main psychodynamic motivation may be de-emphasized, when circumstances do not permit its treatment or constructive exploration, and the assets of the situation pointed up to support or re-establish ego strength. In this connection, I think particularly of the case of an aerial gunner in the last war who inflicted a wound on himself after five rather harrowing combat missions, and who apparently immediately regretted it. I convinced him that although I had sensed the truth, still I did not regard it as anything that interfered with his standing and honor, implying that acts of impulse may also be accidents. He responded to this bit of ego supporting, in which the emphasis was shifted from the past to the future, returned to full duty and completed thirty more missions without recurrence of further medical or administrative disability.

5. Interpretation. Interpretation of any illness to the patient is an important part of treatment. Although I refer here mainly to interpretation of emotional illness, I believe interpretation is necessary for physical illnesses also. Interpretation is the most difficult part of psychotherapy, requiring the greatest amount of skill, tact, and timing. It should be attempted only when one is very sure of his ground and only after a good

relationship has been established with the patient. The essential thing then is to phrase the interpretation in terms acceptable to the patient, and to do this, a genuine understanding of the patient is prerequisite.

Interpretation involves a slow process of understanding and results from the continued cooperative effort of psychiatrist and patient. Results are seldom dramatic, although there are occasional instances where a dramatic denouement may be achieved by a well timed interpretation. Often rather striking results may be obtained by interpreting a patient's hostile wishes or his fears based on his own hostility as reactions to the hate and aggression that he has been subjected to by others in the past or at present. Not so readily acceptable, on the other hand, although often equally true, is the interpretation of anxiety and self-consciousness in terms of projection of repressed aggressive wishes.

Obviously the relation of the interpretation to the self-picture operates here: in the first interpretation the patient is offered good and acceptable reasons for his attitudes and this may help relieve him of considerable guilt; in the second he is faced with some rather unpleasant facts about himself. Such interpretations require an intimate knowledge of the patient's personality and history as well as a good deal of technical experience. They should be made only with the greatest care and only after the patient has fully accepted the therapist and realizes that the therapist in turn has fully accepted him and passes no moral judgment on him. Thus in certain phases of insight-promoting treatment the patient is not supported or built up, but is instead broken down or reduced as he gives up his last-ditch defenses against recognizing trends in himself that are socially unacceptable and deeply disturbing. In certain obsessive anxiety states, for instance, it is especially important to bring about growing insight in which such losing battles and resulting realizations are unavoidable. Experience is needed to carry the patient through these phases of treatment successfully, and supportive phases must be interpolated in order to make the treatment program tolerable as well as constructive and helpful.

Here again is a certain parallelism with age-old ritual practices. This two-phase pattern of treatment—downbeat, uplift—runs like a scarlet thread through all systems of psychotherapy and through much religious practice as was pointed out by Sargant.¹⁴ If this treatment pattern is viewed in Pavlovian terms, one cannot help feeling that it may be the sort of thing Pavlov found necessary to do in treating states of excitation in those animals with "strong but unbalanced nervous systems" who must first be "reduced" or "broken," then built up.

In this limited sense, it may make little difference what method is used to "reduce" the person and to build him up: whether, as in the preliminary phases of religious conversion, a person is reduced by making him conscious of his sinfulness and then built up by infusing him with a feeling of redemption and grace; whether, as in psychoanalysis, he is made conscious of his irrational infantile strivings, then given a feeling of having achieved maturity and insight; whether one disorganizes him temporarily by deep sedation with barbiturates or by insulin coma, then permits him to wake from the prolonged narcosis or insulin coma; whether he sinks to the gutter in chronic alcoholism and is revived and given salvation and status in Alcoholics Anonymous; or whether as in the recent fad of dianetics, he is first convinced that he is nothing but a bundle of mechanical engrams, and

is later elevated to the enviable status of a "clear." In all these ways, first an unsatisfactory behavior pattern is disrupted and disorganized, then the person is helped to build up a new and often better life pattern.

In insight-promoting treatment, actions and attitudes continue to be a powerful instrument of therapy. As in all therapy that offers or suggests insight, however, actions and attitudes if they serve to promote insight will be resisted by defenses to which the patient will cling until he becomes strong enough to give them up; and even when insight is achieved, the symptoms and defenses usually maintain a rebounding pattern of response for a while, with temporary, short-lived recrudescence and often intensification as the immediate response. In other words, if a man is faced with a new idea or principle that momentarily disorganizes his old dynamic integration, not only may the new experience appear to have made no impression, but it may actually result in a less adequate response than before treatment was initiated. Eventually, however, the meaning of the new experience penetrates and can then be integrated into the total behavior of the person. This pattern also finds an analogue in Pavlov's laws of the higher nervous activity of the cerebral cortex, by which the initial negative reaction could be described as negative induction by a new stimulus, followed by positive induction that reinforces the stimulus sufficiently to allow it to irradiate.

6. Suggestion. Suggestion is far less powerful, especially in the severe neurotic illnesses, than is commonly assumed. From a practical standpoint its effectiveness is almost entirely limited to the field of hysteria and to its use as an adjunct to the supportive treatment of psychotic conditions during and after a course of shock treatment, in which suggestibility is greatly enhanced.

Suggestion should be used with greatest care and only after the physician is sure he can produce the suggested effect, which, in practice, is pretty much limited to hysterical states, and then only when the setting has been prepared and the essential conflict worked out. No matter how expertly administered, the power of suggestion should not be used indiscriminately. If a patient of limited intelligence and enhanced suggestibility is overwhelmed by suggestion without sufficient attention to his basic problem, the underlying personality disturbance may well be aggravated by the precipitous taking away of the defensive symptom.

We must always find out what function the symptom serves. This function is sometimes a direct one, sometimes a symbolic one. The symbolism may be translucent, particularly in children whose defenses are lower than those of adults. Suggesting a symptom away by frightening or bossing the child into giving up his symptom is not a constructive way to solve the problem. The symptom itself may be merely a defense. It is therefore exceedingly important to understand the meaning of the symptom before dealing with it in any direct manner. This applies also to other behavior difficulties, particularly in children, in whom the true meaning of the symptom is not difficult to recognize, as well as to psychosomatic disorders, when the underlying conflict must be recognized, rather than merely applying palliative treatment.

The child, for instance, who repeatedly sets fire to the family house more often than not comes from a home with an atmosphere full of hate that includes a state of war between father and mother. To such a child

arson will seem a remedy, radical and drastic to be sure, but not necessarily illogical. After listening to such a child, I try to improve the relationship between the parents. When the strife in the home ends, the child will give up his obvious protest reaction. Any attempt to deal with the protest alone without understanding its meaning would fail. Once the need for the symptom has been abolished or significantly diminished, then suggestion is frequently highly effective as the crowning conclusion of the treatment, but before this, the stage must be set and the patient prepared. An exception may be made in some cases of deeply ingrained hysteria where relief from a symptom by suggestive measures—for instance, hypnosis—may be used as an opening gun in treatment, to be followed by clarification, if not insight therapy, ventilation and support.

From the standpoint of technique, the therapist himself should be convinced that the suggestion will be effective and should be careful not to appear discouraged if it does not take effect immediately. I have found in hysterical states that suggestion sometimes does not attain its full effect until twenty-four hours later, even when hypnosis has been used.

The suggestion should be couched in positive rather than negative terms. It is almost as impossible to obey the command, "Don't worry," as an instruction not to think of a black-and-white speckled bear. Such positive suggestions as "Be confident and optimistic" and "You will be happy, well, and strong" carry weight in the right direction. The same applies to the formulation of posthypnotic suggestions. The formulation, "You will have no headaches," is far less effective than "Your head will feel clear and well."

In suggestion we apparently appeal to categories of thought, so that the relatively colorless negative prefixes and adverbs have little force. In a study of fires aboard ship, a shipping company found the inscription "In case of fire do not open this door" carried far less weight than the subsequent version "In case of fire keep this door shut." Salesmen know that the slogan "Our radiators do not leak," no matter how truthful, would sell few radiators; psychologically the idea of leaking remains, while the colorless "not" vanishes.

7. Persuasion. Persuasion as a specific tool of psychotherapy was introduced by Dubois^{6,7} and Déjérine,⁵ but its use is probably much older. Today persuasion has been resolutely abandoned, at least consciously, by modern psychotherapists. Nevertheless, it still continues to be practiced, albeit in a large part unconsciously and by implication when it often boils down to the old battle of wits between patient and doctor.

Even those modern psychotherapists who warn against its authoritarian implications still must resort to it on occasion, although they do so tactfully and with subtlety in contrast to its previous more direct use. Frieda Fromm-Reichmann, whom I among others consider one of our best psychotherapists and an excellent representative of the psychodynamic approach, states, for instance:⁹ "The psychiatrist should never feel as if he were a clever detective outwitting the culprit in a simple maneuver. Instead, he should make a point of *instigating the patient's interest* in cleverly discovering for himself the hidden meaning of his intricate communications." (pp. 127–128; italics mine.) This "instigation" which Dr. Fromm-Reichmann recommends is of course persuasion, and to bring it about the patient

must be convinced of the validity of the procedure the therapist wants the patient to carry out with him.

Dr. Fromm-Reichmann goes even further when she states, "The psychiatrist should not be afraid to present correct and valid interpretations with the authority of a well-trained person who is certain that he knows his business. His formulations should be short, simply and plainly meaningful, and in the vein of matter-of-fact suggested leads offered by one person, the therapist, to another human being, the patient, who needs guidance" (p. 134). Bringing this about again emphasizes the importance of the therapist's victory in the battle of wits, although of course he must carry out this battle with a velvet glove.

The element of battle in a modern sense of a resourceful war of movement is further emphasized by Dr. Fromm-Reichmann's recommendation of flexibility: "The formulations which the psychiatrist uses with each patient should vary according to the type of personality, the particular psychopathology, and the special background of the patient in question" (p. 135). Compliance with these rules will facilitate the patient's acceptance of an interpretive suggestion. She also recommends: "In the case of patients who persist in a refractory attitude toward verbalized routine interpretive work or in the case of an emergency, the psychiatrist should use his ingenuity to expedite the situation. He must call upon those devices which are available to him in accordance with his skill and experience and which are contingent upon his own and his patient's personalities" (p. 135).

Among the devices Dr. Fromm-Reichmann uses are interpretations of the physical symptoms in terms of meaningful underlying attitudes that the patient can actually feel and thereby accept, such as, for instance, when she gave a patient with a *tic douloureux* insight into its relation to her general attitude of sneering disdain.

From the heights of our present psychodynamic understanding the devices used by therapists in the old days seem clumsy and often absurd; yet we must admit they have certain basic features in common with our present methods. Dubois, for example, explained things to patients by using the working of the steam engine and its pistons as an analogue. His interpretations were mechanistic, yet in a vast number of cases they seem to have worked almost as well as our present highly sophisticated psychodynamic explanations. The same holds with certain other old devices. Sir John Bland-Sutton⁴ tells the following story (p. 59): "A thin and foolish woman believed she had accidentally swallowed a frog and that her thinness was due to the frog eating the food in her stomach. In order to dispel the illusion the doctor gave the patient an emetic and during the vomiting he slipped a small frog into the basin. When the patient saw the frog her joy was great, but in a few minutes her depression returned: 'Oh!' she exclaimed, 'I am sure this frog has left some young ones in my stomach.' The doctor looked wise, pulled out his magnifying glass, and after critically examining the frog, said unto the patient, 'Fear not; this frog has not left any froglets inside you. Behold, it is a male!' The patient was quite satisfied, became happy, and in a few months was plump again. She was not a naturalist, and therefore ignorant of the fact that it is difficult to tell the sex of frogs by mere inspection except at the breeding season."

Today we would probably have solved the problem by explaining to the woman that the frog represented a pregnancy fantasy and that she was in a

arson will seem a remedy, radical and drastic to be sure, but not necessarily illogical. After listening to such a child, I try to improve the relationship between the parents. When the strife in the home ends, the child will give up his obvious protest reaction. Any attempt to deal with the protest alone without understanding its meaning would fail. Once the need for the symptom has been abolished or significantly diminished, then suggestion is frequently highly effective as the crowning conclusion of the treatment, but before this, the stage must be set and the patient prepared. An exception may be made in some cases of deeply ingrained hysteria where relief from a symptom by suggestive measures—for instance, hypnosis—may be used as an opening gun in treatment, to be followed by clarification, if not insight therapy, ventilation and support.

From the standpoint of technique, the therapist himself should be convinced that the suggestion will be effective and should be careful not to appear discouraged if it does not take effect immediately. I have found in hysterical states that suggestion sometimes does not attain its full effect until twenty-four hours later, even when hypnosis has been used.

The suggestion should be couched in positive rather than negative terms. It is almost as impossible to obey the command, "Don't worry," as an instruction not to think of a black-and-white speckled bear. Such positive suggestions as "Be confident and optimistic" and "You will be happy, well, and strong" carry weight in the right direction. The same applies to the formulation of posthypnotic suggestions. The formulation, "You will have no headaches," is far less effective than "Your head will feel clear and well."

In suggestion we apparently appeal to categories of thought, so that the relatively colorless negative prefixes and adverbs have little force. In a study of fires aboard ship, a shipping company found the inscription "In case of fire do not open this door" carried far less weight than the subsequent version "In case of fire keep this door shut." Salesmen know that the slogan "Our radiators do not leak," no matter how truthful, would sell few radiators; psychologically the idea of leaking remains, while the colorless "not" vanishes.

7. Persuasion. Persuasion as a specific tool of psychotherapy was introduced by Dubois⁶,⁷ and Déjérine,⁵ but its use is probably much older. Today persuasion has been resolutely abandoned, at least consciously, by modern psychotherapists. Nevertheless, it still continues to be practiced, albeit in a large part unconsciously and by implication when it often boils down to the old battle of wits between patient and doctor.

Even those modern psychotherapists who warn against its authoritarian implications still must resort to it on occasion, although they do so tactfully and with subtlety in contrast to its previous more direct use. Frieda Fromm-Reichmann, whom I among others consider one of our best psychotherapists and an excellent representative of the psychodynamic approach, states, for instance:⁹ "The psychiatrist should never feel as if he were a clever detective outwitting the culprit in a simple maneuver. Instead, he should make a point of *instigating the patient's interest* in cleverly discovering for himself the hidden meaning of his intricate communications." (pp. 127-128; italics mine.) This "instigation" which Dr. Fromm-Reichmann recommends is of course persuasion, and to bring it about the patient

must be convinced of the validity of the procedure the therapist wants the patient to carry out with him.

Dr. Fromm-Reichmann goes even further when she states, "The psychiatrist should not be afraid to present correct and valid interpretations with the authority of a well-trained person who is certain that he knows his business. His formulations should be short, simply and plainly meaningful, and in the vein of matter-of-fact suggested leads offered by one person, the therapist, to another human being, the patient, who needs guidance" (p. 134). Bringing this about again emphasizes the importance of the therapist's victory in the battle of wits, although of course he must carry out this battle with a velvet glove.

The element of battle in a modern sense of a resourceful war of movement is further emphasized by Dr. Fromm-Reichmann's recommendation of flexibility: "The formulations which the psychiatrist uses with each patient should vary according to the type of personality, the particular psychopathology, and the special background of the patient in question" (p. 135). Compliance with these rules will facilitate the patient's acceptance of an interpretive suggestion. She also recommends: "In the case of patients who persist in a refractory attitude toward verbalized routine interpretive work or in the case of an emergency, the psychiatrist should use his ingenuity to expedite the situation. He must call upon those devices which are available to him in accordance with his skill and experience and which are contingent upon his own and his patient's personalities" (p. 135).

Among the devices Dr. Fromm-Reichmann uses are interpretations of physical symptoms in terms of meaningful underlying attitudes that the patient can actually feel and thereby accept, such as, for instance, when she gave a patient with a *tic douloureux* insight into its relation to her general attitude of sneering disdain.

From the heights of our present psychodynamic understanding the devices used by therapists in the old days seem clumsy and often absurd; yet we must admit they have certain basic features in common with our present methods. Dubois, for example, explained things to patients by using the working of the steam engine and its pistons as an analogue. His interpretations were mechanistic, yet in a vast number of cases they seem to have worked almost as well as our present highly sophisticated psychodynamic explanations. The same holds with certain other old devices. Sir John Bland-Sutton⁴ tells the following story (p. 59): "A thin and foolish woman believed she had accidentally swallowed a frog and that her thinness was due to the frog eating the food in her stomach. In order to dispel the illusion the doctor gave the patient an emetic and during the vomiting he slipped a small frog into the basin. When the patient saw the frog her joy was great, but in a few minutes her depression returned: 'Oh!' she exclaimed, 'I am sure this frog has left some young ones in my stomach.' The doctor looked wise, pulled out his magnifying glass, and after critically examining the frog, said unto the patient, 'Fear not; this frog has not left any froglets inside you. Behold, it is a male!' The patient was quite satisfied, became happy, and in a few months was plump again. She was not a naturalist, and therefore ignorant of the fact that it is difficult to tell the sex of frogs by mere inspection except at the breeding season."

Today we would probably have solved the problem by explaining to the woman that the frog represented a pregnancy fantasy and that she was in a

hostile manner devaluating herself and her potential offspring by degrading it to a frog. Further genetic explanation of this fantasy might go back to early and exceedingly complex roots involving still highly speculative theory. But whether such a complex psychodynamic interpretation would, from her point of view, have much more reality in reference to her problem than what the eager doctor slipped into her vomitus is highly debatable.

In a like vein, it is of interest that the witch doctors among the natives in Kacongo also treated bewitched or insane persons by massage and an emetic to remove the demon, whereupon the witch doctor saw to it that the disturbed person always found several lively young crocodiles in his vomitus (according to an account by Mary Kingsley quoted in Sir John Bland-Sutton⁴).

Presenting a patient with a frog or crocodile introduced by sleight of hand (a practice nowadays justly frowned upon) and a psychodynamic explanation conjured up by means of scientific ingenuity (a practice nowadays highly regarded) while seemingly two highly divergent approaches, have these two points in common: they convince the patient that there is substance and reality to his complaint and that the method he is being treated by is capable of abolishing the cause of his misery.

These graphic and primitive methods were of course adapted to the primitiveness of the patients, thus conforming to the psychotherapeutic axiom that interpretation must be made in terms acceptable to the patient. It appears that giving the patient the conviction that what ails him has been amply explained and can be effectively done away with is somehow a strong element in all these techniques. In spite of the greater scientific validity of our present interpretations, still inherent in our psychotherapy is that basic element of persuasion, of convincing the patient we have adequate control over and understanding of cause.

These observations are further highlighted by the fact that the effect of an interpretation is not always correlated with its validity. Frieda Fromm-Reichmann expresses the problem succinctly: "Under the pressure of the expectations of the psychiatrist and because of the eagerness of their dependent and suffering patients to comply and to please, patients may frequently produce what their psychiatrists are looking for. However, that is no proof of the correctness of these interpretations; but it evidences the innate competence of human nature. It is due to this competence that some mental patients are capable of using the helpful dynamisms inherent in all psychotherapeutic exchange for the benefit of their striving toward recovery. It may be said that this sometimes does happen regardless of the validity of the interpretation" (pp. 84-85).

As in the battle of arms, in the battle of wits the ends sometimes glorify the means.

8. Hypnosis. A good many elements of psychotherapy are combined in hypnosis, including ventilation, abreaction, suggestion, persuasion, and insight therapy. Hypnosis is a state of reduced wakefulness brought about in the patient by his co-operation with the directions given him by the therapist.² Its beneficial effects are associated with the following four factors:

(a) An increased awareness of psychosomatic relationships which impresses itself particularly strongly upon the patient in the initial stages of hypnosis; its effects are akin to those of the Yoga methods of Far Eastern faith healers. This increased awareness—for instance, after concentration on warmth and heaviness, the consciousness of the increasing

"weight" of an extremity produced by hyperemia and relaxed tonus—is particularly beneficial in anxiety states and hypochondriasis.

(b) Increased suggestibility, which appears at a somewhat deeper stage of the hypnosis and enables the patient, under the impact of suggestion, to break through the emotional blocking of certain functions. This aspect of hypnotic therapy is especially important in hysteria and related conditions in which its success in eliminating the distressing symptoms renders the patient more accessible to subsequent psychotherapy directed at more basic aspects of the disorder.

(c) Catharsis. When the so-called somnambulatory stage of hypnosis is reached the patient is ready to talk, or at least to answer questions, about the basic psychologic factors operative in his illness which he either represses or is only partially aware of in the waking state. The clarity of formulation that many not particularly eloquent or clear-thinking patients are capable of in this stage of hypnosis is often extraordinary and can be compared only with the directness and clarity of emotional expression in certain dreams.

The therapist should follow up the catharsis with equally direct and clear-cut psychotherapeutic directives concerned with interpretation, persuasion, and strong suggestion, the last particularly in reference to release from subjective guilt feelings. In this stage of hypnosis the acceptance of intensive psychotherapy by certain patients is so greatly enhanced that the results of one session often equal or surpass those obtained by weeks or months of painstaking psychotherapy in the waking state. This cathartic-psychotherapeutic aspect of hypnotic treatment is particularly helpful in anxiety-tension states, obsessive states, anxiety neurosis, and reactive depression.

(d) Activation. It is well known that in hypnosis a subject may demonstrate greater physical strength than he can in the waking state, probably because of the greater concentration possible in this state whereby more motor units can be brought into simultaneous play in the same muscles than is usual in ordinary voluntary motor innervation. The subjective experiencing of this extraordinary motor strength, especially if it occurs in the lighter stages of hypnosis, has a pronounced activating effect on the patient. If this is carried over into the waking state by appropriate post-hypnotic suggestion, it can be useful in treating many conditions associated with inhibition or reduction of impulse—even, temporarily, in those caused by organic cerebrospinal disease.

The physiologic aspects of hypnosis are too complicated for the scope of this book. The reader is therefore referred to the literature and standard texts on this subject, particularly to Wolberg.^{16, 17}

9. Reassurance. Reassurance may be regarded as part of supportive treatment, but because of its specific nature it should not be used until a general groundwork of other supportive measures has been solidly established. As mentioned before, reassurance like suggestion must be given with great caution. For reassurance to be effective, it is essential that the patient believe the doctor is truthful and that he has been sufficiently thorough and has sufficient knowledge to know whereof he speaks. In taking care of a patient through an illness, the doctor's role is very much like that of an Indian guide taking a wayfarer through the wilderness. Such a traveler has heard of sand-traps, hidden swamps, hostile Indian tribes, and

rattlers in hiding. Trying to find his way through all these hazards alone would be a harrowing and frequently frightening experience. But with a good Indian guide, known to have taken others through safely, the journey could be undertaken with confidence.

In psychoses or severe neuroses, reassurance is of only secondary importance apart from its use as an adjunct to general supportive treatment. Reassurance may, however, be of great importance as a preventive measure and is particularly important in the very essential psychotherapy of patients suffering from incurable or chronic physical illnesses, just as it is with patients in any stressful situation which cannot be changed. In these conditions a great deal depends on the physician's psychotherapeutic handling of the problem. The existence of such an illness constitutes severe neurosis-producing stress, and it is frequently this superimposed neurosis or depression which makes the difference between an invalid and a reasonably active, well adjusted individual. The manner in which the physician handles such a problem may prevent, control, or on the other hand, even precipitate this superimposed neurosis. Thorough warm understanding of the patient combined with a knowledge of his emotional needs will do much to prevent such superimposed neurotic reactions. At the same time it is possible, indeed imperative, for the physician to be honest with the patient and allow him to continue to square his condition with reality, that is, permit him to retain reality testing along with other essential ego personality supports. Therefore I favor giving the patient the true diagnosis, but always in encouraging terms. Withholding the diagnosis sometimes makes the patient fear an even worse possibility than that which has been well-meaningly withheld.

An extremely important point in maintaining ego strength is to maintain in the patient a feeling of control, a feeling that he can do something for himself, that he is not entirely helpless. Abandonment to Fate constitutes extreme stress which few but the most sturdy can take. In combat personnel in war, for instance, we found that subjective flying stress and resulting neurosis were far less frequent in fighter pilots who in combat could maneuver freely on their own decision than it was in bomber pilots whose course was predetermined from the starting point to the target, regardless of opposition encountered, the bomber pilot being unable to make any maneuvering action of his own in this crucial part of his flight.

One should therefore never tell any patient, "There is nothing I can do for you." without at least offering a program of things he can do for himself and continuing to show a helpful interest. All too frequently such patients are abandoned to their own devices with the pessimistic implications uppermost. The rationalization for doing this is usually a completely ethical one. The real motive, however, is more complicated. The chronically or incurably ill patient represents a tremendous challenge to the physician's self-esteem regarding his healing abilities, which will frequently put him on the defensive, especially when the patient on his part enhances this challenge by openly hostile, demanding, or critical attitudes. It is therefore understandable that in such a setting the doctor, especially when he is sensitive to criticism and hostility, may tell the patient he can do nothing for him in such a way as to imply, "There is nothing *anybody* can do for you." This to the patient means complete rejection and abandonment. We must never forget that a principal function of the

physician is to give hope to the patient and to relieve his relatives of responsibility by not dodging it himself.

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VII

GENERAL PRINCIPLES OF TREATMENT BY SHOCK, STIMULATION, AND PSYCHOSURGERY

INSULIN TREATMENT

Modern physical treatment of the psychoses had its origin in the early 1930's, starting with a method developed by Sakel for treating morphine addiction.⁷¹ Sakel began using injections of insulin in subcoma doses in treating the symptoms arising from abstinence in morphine addicts, on the theory that insulin might influence the functioning of the nerve cells in such a way as to relieve the patient's excitement. When such insulin treatment succeeded in decreasing the distressing symptoms of the newly abstinent morphine addict, Sakel decided to try it for treating other forms of excitation, in the hope of being able to tone them down too. Quite accidentally, when he attempted to determine optimal dosage, some of the schizophrenic patients being treated fell into coma. When they emerged from coma, Sakel found that not only had the excitement abated but the psychotic symptoms themselves had lessened and in some cases even disappeared. Thus, insulin coma treatment was born, and in it the most successful form of therapy of mental illness that had yet been found. The rationale, other than that based on its empiric value, was then and still is to a large extent obscure.^{72, 73, 74, 75}

METRAZOL TREATMENT

Three years later Meduna⁶² introduced a method of inducing convulsion by the injection of metrazol. His rationale was based on the erroneous belief that epilepsy and schizophrenia do not coexist in the same person and might, in fact, be antagonistic to each other. He was probably also influenced by the fact that insulin-treated schizophrenics occasionally go into convulsions, following which they sometimes make more rapid progress toward recovery. Since then this theoretical rationale has been quite thoroughly demolished. It has been established that epilepsy may occur in schizophrenia, and in my own experience, as well as that of other clinicians, spontaneous convulsions in schizophrenics have no therapeutic benefit whatsoever in relation to their psychosis. It was also soon discovered (Bennett¹¹) that convulsive treatment is of far more striking benefit in manic-depressive psychosis than in schizophrenia. Furthermore, convulsive treatment was found to be of benefit even in epileptics (Taylor⁸⁹).

ELECTROSHOCK TREATMENT

Starting from E. A. Spiegel's⁷⁹ discovery that convulsions in animals can be readily produced by passage of electric current through the brain and that the convulsions so produced do no apparent harm to the animal—a method which had also been successfully used by Putnam and Merritt^{63, 69} to test the efficacy of anticonvulsant medication—Cerletti and Bini²⁰ introduced a method of cerebral electric stimulation as a means of bringing about therapeutic convulsive seizures. Their method rapidly became popular throughout the psychiatric world and soon entirely replaced metrazol, for as soon as the electric current was applied the patient was rendered immediately unconscious in contrast to the period of waiting for the convulsion after injection of metrazol, a period that was tense and anxiety-provoking for both patient and physician.

As use of the new method spread, it was soon found that the conditions responding most favorably to such treatment were the affective psychoses rather than the classic schizophrenic ones, although certain psychotic conditions in which affective and schizophrenic features commingled, such as the catatonic state, also seemed to benefit.

Certain drawbacks of the electrical method of producing convulsions became evident at times in disturbing side effects, predominant among which were a marked confusion and apparent memory loss, both of seeming "organic type," occasional alarming respiratory and circulatory side effects, and, in a significant number of cases, marked build-up of anxiety and aversion to treatment on the part of the patient in spite of his experience of relief from the depression and other manifestations of the psychosis. The confusion and apparent memory disturbance were so similar to those found in organic psychoses and deterioration that the possibility of organic brain damage had to be seriously considered, and although well controlled studies in animals failed to reveal any irreversibility of changes in the brain, the possibility that this might merely reflect inadequacy of our neurohistologic methods could not be overlooked. Some workers even theorized that the marked organic confusion and memory defect might in themselves comprise the therapeutically beneficial elements of the treatment. In consequence, the fear that brain damage might result has interfered with general acceptance of the method in spite of its great therapeutic efficacy.

At the same time, the build-up of postshock anxiety remained entirely unexplained. Cerletti himself, as late as 1950,¹⁹ suggested that it was due to unconscious anguish experienced during the treatment that later emerged into consciousness, a theory entirely contradicted by the apparently organic amnesia and confusion.

In the course of research carried out for the specific purpose of the elucidation of the role of these factors, I have arrived at a quite different viewpoint (as will be discussed in greater detail in later chapters). Briefly, on the basis of this research, it seems most probable that most of the organoid changes seen following electroshock treatment are due to temporary physiologic inhibition (in Pavlov's sense), rather than to organic deterioration, that postshock anxiety is also a physiologic response, and that both side effects can be either eliminated by subsequent nonconvulsive treatment or prevented by following the convulsive phase immediately by

nonconvulsive treatment. Funkenstein and his coworkers have shown that postshock anxiety is associated with an increase in adrenergic responsiveness; it occurs only in those patients in whom anxiety can be precipitated by epinephrine, a reaction which can be demonstrated before treatment. In subsequent collaboration with Funkenstein I have found that nonconvulsive electric treatment lowers and/or abolishes such epinephrine-precipitable anxiety.

Treatment with Low Amperage Unidirectional Currents. The first workers who made a determined effort to prevent the side effects of electroshock treatment, in the belief that they were not related to its therapeutic effects, were Friedman, Wilcox, and Reiter³³ in this country and Delmas-Marsalet^{22, 23, 24} in France. These workers believed the side effects were due to the unphysiologic aspects and current patterns of the massive alternating currents used in electroshock treatment, and they substituted unidirectional current of longer duration and far less amperage.

The first Reiter Electrostimulator, on which Wilcox and Friedman collaborated with Reiter, an electrical engineer, utilized pulsating direct current with 60 half-sine waves per second, modified by a condenser which produced a steep wave front and a gradual wave decline. In 1940, experiments were initiated by them with varying patterns of stimulation to determine the efficacy of brief, lengthy, and steady stimulation as well as the effect of gradual increases and decreases in the strength of the stimulation.³³

These men also developed an electrode vertex-temporal position which has since been shown to require less current than the bitemporal position widely used in standard electroshock treatment (Gottesfeld, Lesse and Herskovitz⁴⁰). The average general output of current required to produce a convulsion with this machine was 40 milliamperes applied for a total period of from 1 to 2 seconds. The machine also employed a mechanical interrupter which Reiter introduced into the output circuit, and this procedure obtained successive pulsations, followed by brief pauses, which tended to break up the patient's motor thrust and initiate the convulsion in a more gradual manner than was possible with the Cerletti-Bini technique. Obviously, the incidence of fracture was thus lowered; also there was less frequent appearance of organoid brain syndromes. However, there was an increased evidence of apprehension with the early Reiter technique compared to the Cerletti-Bini method.

In 1945 Liberson^{52, 55} undertook experiments with a new "brief stimulus" current he had utilized in a successful effort to demonstrate that less memory loss was experienced with his technique than with the Reiter machine. Physiologic analysis of the excitabilities involved in the stimulation of the cortical motor neurons showed that their chronaxies are similar to those of peripheral motor nerves, which vary between 0.08 and 0.7 millisecond. When the duration of electrical pulses is equal to or below one chronaxie of the corresponding tissue, stimulation will be obtained with the lowest electrical energy possible. The duration of each pulse of the stimulating current is much longer than the chronaxie of the cortical motor neurons (Medlicott⁶⁰). It was Liberson's intention to prove that by lowering the duration of the pulses, convulsions could be produced with minimal electrical energy, with the attendant advantages of reduced hazard of

brain damage, intellectual impairment, and memory loss. On the other hand, the Liberson method produced even more apprehension of treatment than the Wilcox-Friedman-Reiter technique.

In 1946 a new brief stimulus machine was constructed by Liberson,^{53, 54} incorporating a resistance-estimating device which automatically regulated the voltage in accordance with changes in the patient's body resistance. Liberson then introduced the idea of further modifying the unidirectional current employed to produce seizures with still less electrical energy, employing about 120 impulses per second with brief waves approaching "square waves" in form, and with a total duration of stimulation averaging 1 second. An average stimulation adequate to produce convulsion consisted of a current of 350 peak milliamperes with an individual pulse duration of 0.2 to 0.5 millisecond flowing for 0.5 to 1.0 second.

It soon became apparent that the Liberson technique was hedged about with restrictions, in spite of the low energy equations. The awareness threshold failed to coincide with the convulsive threshold, and convulsions induced with low pulse peaks were often accompanied by awareness of painful sensation, occasionally a glimmering of awareness of the convulsion itself. When higher pulse peaks were used, the risk of fracture was increased as the motor thrust was increased. Nevertheless, the brief stimuli technique presented modifications of the major objectionable features of standard domestic alternating current; the threshold convulsive dose using unidirectional current was appreciably lowered, and, with it, the incidence of memory disturbance, confusion syndromes, EEG changes, while the feasibility of lengthened treatment series was greatly enhanced. "In older people, where numerous treatments are at times necessary, sufficient treatment can be given with one BST machine to produce adequate benefit, when treatment with the classic type of current would have to be abandoned because of its extreme effect on the state of consciousness." (Goldman.³⁸) The additional feature of increased frequency and shorter total stimulation time in the brief stimulus method reduced the anxiety which accrues when longer times of stimulation are used.

In 1948 Medlicott in New Zealand reported a high degree of success with the brief stimuli technique, particularly in regard to a lesser degree of mental and emotional dulling following treatment, as compared to standard electroshock treatment. He found it possible to keep better contact with brief stimuli treated patients and to give them more adequate psychology help during the treatment series. He also corroborated other reports that there appears to be no difference in recovery rates between the standard electroshock treatment and brief stimuli treated patients (Medlicott⁶¹).

In 1946 a new Reiter machine was developed (Wilcox⁹⁵) which employed a series of sharp-spiked unidirectional current pulses with scattered maximum peaks, constituting an even smaller portion of the total energy than the previous square-topped pulses did; only 6 average d.c. milliamperes for 2 to 3 seconds were required for a threshold convulsion. Even more gradual onset of the convulsion was thus obtainable, further reducing the fracture hazard, and also producing milder tonic and clonic phases. With the standard electroshock treatment method, frequently a pause of several seconds (6 to 70) elapsed following the production of a fit before respiration was resumed. The new Reiter apparatus reduced apnea to a

obtained electronarcotic effects with alternating currents.⁸⁸ In 1942 Van Harreveld and his colleagues⁸⁹ observed that direct current of sufficient intensity has a much weaker narcotic effect than pulsating or alternating current, because, owing to accommodation and resulting tolerance, possibly an effect of polarization, its stimulating power is much smaller. Nevertheless, any variation in current of sufficient duration and slope can be used to effect electronarcosis. The authors at this time advanced the hypothesis that when diffuse application of current to large parts of the central nervous system—as in the production of electronarcosis—stimulates structures with excitatory as well as those with inhibitory function, inhibition prevails over excitation; according to this view, the normal central inhibition is responsible for electronarcosis.

They also noted that the symptoms of electronarcosis produced in dogs could develop in either of two directions, which they designated as “narcotic”—characterized by a quiet, relaxed reaction—or “kinetic”—a more active state, with some violent hyperkinetic action. The type of reaction obtained was an individual matter, varying from one animal to the next.

Shortly thereafter, Globus et al.,³⁷ in their studies of dog brains which had undergone electronarcosis, reported that they could find no significant changes either with gross or microscopic examination, thus lending support to the growing acceptance of electronarcosis as a safer form of therapy. In 1944 Frostig and his coworkers³⁴ studied animal and human reactions to electronarcosis. They emphasized that a fundamental difference exists between chemically and electrically produced narcosis, since the narcotic action of the electric current, as was shown by Van Harreveld et al.,⁸⁹ depends on its stimulating effect. The metabolism of the brain, which is reduced by chemical narcosis, remains practically unchanged during electronarcosis.⁸¹ The reactions which they observed in the 9 human patients—all schizophrenics of long standing—were a combination of the narcotic and kinetic types of reactions observed in their experiments with dogs. They used an initial current of 150 to 250 milliamperes for 30 seconds, then decreased the current to a point at which respiration commenced (45 to 90 milliamperes). It was found that the current must then be gradually increased to maintain an electronarcotic level, as tolerance to the amount of current gradually develops. Termination of electronarcosis was indicated, they felt, as soon as the appearance of intentional movements revealed the resumption of cortical activity. Cutting off the current resulted in prompt return to consciousness; no patient reported pain or other unpleasant effect.

Various electrode placements were tried, but electronarcosis was found to remain constant regardless of placement, except that occipital placement resulted in greater impairment of respiration than did more frontal placements. The heart rate in humans reacted in similar fashion to that of the dogs. After the initial high current had produced cardiac arrest for a few seconds, the heart beat was then resumed at a low rate, in one and a half minutes of treatment reached 150–180 (considerably above the pre-narcotic level), and then began to decrease, often falling below the pre-narcotic level. Three to four minutes after the beginning of the treatment, the heart rate showed another increase to between 120 and 160 which was maintained for the rest of the period of electronarcosis.

Blood pressure similarly dropped with the first application of a high

current, then rose with the heart action to about the prenarctic level, although it often went higher, up to 180 to 200 mm. systolic. However, the authors could draw no conclusions from the individual variations. Medlicott⁵⁹ reported a gradual rise in blood pressures, occasionally attaining a hypertensive level, and Goldman³⁹ reported a similar rise, but did not consider such increases dangerous unless there was advanced arteriosclerosis.

In dogs, the blood sugar often more than doubled during electro-narcosis. This same rise was noted even when the initial cardiac arrest—which might be thought responsible—was prevented by the use of atropine. The authors suggested that the blood sugar rise was probably due to the stimulation of the sympathetic system. A material increase was also noted in red and white corpuscles of the blood during electro-narcosis.

Method of Induction. The accepted method of induction of electro-narcosis was devised by Tietz,⁸⁵ who in 1947 added a new precautionary measure of current application, termed by her and her coworkers⁸⁴ the "glissando" technique because of the gradual rate of current increases. Skeletal complications which were often observed following the initial jolt with which the current was applied were now greatly reduced if not completely obviated by raising the current from zero to the treatment level over a period of 1 to 2 seconds. However, the current must reach 150 milliamperes within 2 seconds, or else it will be felt and remembered by the patient.

To induce electro-narcosis an initial current of from 150 to 200 milliamperes is applied for 30 seconds, following which it is decreased to 45 to 90 milliamperes. Respiration commences within 40 to 60 seconds of the start of treatment; one to one and a half minutes after the beginning of treatment, the current is gradually increased, usually 5 milliamperes each 15 seconds to a maximum of 125 milliamperes or until stridor develops. The usual duration of treatment is 7 minutes, although electro-narcosis has been maintained experimentally as long as 25 minutes without ill effect.⁵⁹ Several workers report that they routinely use carbogen (oxygen with 5 per cent carbon dioxide) after the initial high current application, and maintain its use throughout treatment. Tietz⁸² also uses curare routinely (1.5 cc. intravenously two minutes prior to treatment), and all workers use sodium amytal or pentothal either routinely or when they feel its use is indicated by the apprehensiveness of the patient. Tietz and others also recommend the routine use of vitamin therapy throughout the treatment series.

The initial application of a high current in electro-narcosis produces a modified convulsion, that is, a tonic spasm, first of flexor and then of extensor muscles. When the current is reduced, a few clonic movements may be observed, succeeded by brief apnea of from 15 to 30 seconds duration. A state of unconsciousness is then maintained by gradual increases in current, either to an arbitrary maximum (usually 125 milliamperes) or until spasms of the extensor muscles of the lower extremities and flexor muscles of the arms are observed. As Karliner⁴⁷ has pointed out, quite often inspiratory stridor develops before this level of the narcotic state can be reached, and this presents one of the most difficult problems in the induction of electro-narcosis, one which, however, can generally be overcome by decreasing the current and then slowly and progressively raising

it again, together with the administration of carbogen (oxygen 95 per cent, carbon dioxide 5 per cent).

Effects. While the initial seizure in electronarcosis bears resemblance to the grand mal convulsion seen in standard electroshock treatment, the period of unconsciousness which ensues in electronarcosis resembles insulin coma in several respects. Many workers^{39, 59, 82} have taken note of the increased stimulation of the autonomic nervous system in electronarcosis, resulting in excessive perspiration, lacrimation, salivation, and pilomotor reaction of the trunk and limbs, as well as gradual flushing of the skin. During insulin coma therapy as well as during electronarcosis, forced grasping and prolonged flexor tone are also observed. However, Tietz and Van Harreveld⁸⁶ have pointed out in their experiments with animals that the large rise in the level of "epinephrine-like" compounds in the blood during electronarcosis is caused by asphyxiation rather than by the current itself. When asphyxia-free electronarcosis was administered to rabbits, only a very slight rise was noted, which they believe is due to the release of epinephrine.

Simon, Bowman, and Halliday⁷⁸ observed that electronarcosis initiates a hyperglycemia which reached a peak of 52 mg. per cent increase over pretreatment levels in 20 minutes, while in patients treated with standard electroshock treatment an increase of 25 mg. per cent was noted after the same interval. They suggest this as evidence of intense sympathico-adrenal stimulation in electronarcosis.

Electroencephalographic changes are less pronounced in electronarcosis than with standard electroshock treatment.³⁹ Much less memory loss follows electronarcosis than follows either brief stimuli treatment or electroshock treatment, whereas apprehensiveness of treatment is much more common than with standard electroshock treatment.

Skeletal complications are extremely rare in electronarcosis; cardiovascular complications have been rare. The coma induced by electronarcosis can be terminated immediately in case of circulatory or respiratory difficulties, an advantage over insulin coma therapy in which glucose must be administered. Fewer personnel and less time per patient are required in electronarcosis as compared with insulin coma therapy. Some patients who are poor risks for continued insulin treatments because of susceptibility to delayed insulin coma, repeated nausea following termination of the comatose state, or other reasons may be more safely treated with electronarcosis.

Results. Electronarcosis has now come to be considered by many psychiatrists as the treatment of choice in schizophrenia; results show it to be superior to electroshock treatment in these cases and comparable to insulin coma. Medlicott⁵⁹ in 1947 treated 50 schizophrenics with electronarcosis and his results corroborate this statement, as do further reports by Pater-son and Milligan⁶⁵ in the same year, and the series of studies done by Tietz in 1945,⁸⁵ 1947,⁸² and 1948.⁸³

Medlicott noted particularly that in paranoid schizophrenics—patients with well developed delusional states and unfavorable prognoses—electronarcosis obtained much better results than standard electroshock treatment, and this author recommends electronarcosis as the therapy of choice for this group. Of 16 paranoids, he reports 37.5 per cent fully recovered, 12.5 per cent partially recovered (social remission), and another 12.5 per

cent improved. Similarly, Tietz⁸² reports that of 18 schizophrenics with paranoid features, 14 effected a full recovery, 3 a social recovery, and only one, with hebephrenic features, showed very little improvement.

Medlicott feels, however, that in acute schizophrenic reaction states and in schizophrenics of undetermined or mixed group, electronarcosis offers no advantage over electroshock treatment. In simple schizophrenia and in schizophrenia with predominantly hebephrenic features, he believes that electronarcosis produces results similar to those obtained with electroshock treatment, but does so in a shorter period of time.

Tietz⁸² found that of a group of 13 psychoneurotics with predominantly depressive features, electronarcosis effected a full recovery in 9 cases and a social recovery in 2. She also obtained excellent results in 7 cases of recurrent depressions, and poorest results with a group of female paranoid involutional psychoses with symptoms of long standing. Most of the female patients in this series developed amenorrhea which continued for a month or two following termination of treatment.

Paterson and Milligan,⁶⁵ working in England, reported enthusiastically on their results with electronarcosis in the treatment of schizophrenia, using a machine in which constant current is obtained without the use of thermionic valves. They regarded their results as far superior to those obtained with other methods. All 22 schizophrenics with hallucinations whom they treated were relieved of this symptom early in the treatment series.

Goldman in 1949³⁹ reported results with 125 patients, who, almost without exception, had previously been subjected to other forms of therapy with little or no improvement, and had been ill for long periods. He considers the most favorable prognostic group for application of electronarcosis to be the affective psychoses, whether with depressive, paranoid, or catatonic features, as well as those psychoneuroses in which compulsive anxiety is a predominant feature. "Apparently, if any one characteristic can be seen which runs through the entire group" (favorably influenced by electronarcosis), "it is the presence of deep anxiety, otherwise unapproachable by any means whatever." Goldman believes that it is this type of anxiety which electronarcosis alleviates in a specific way, unequalled by any other method except lobotomy.

Freund,³¹ in the course of using electronarcosis as an adjunct to psychotherapy, has noted that particularly in patients who had proved beyond contact by any other method, release of traumatic material valuable in subsequent psychotherapy was elicited immediately after electronarcosis.

Tietz, in a further follow-up study of two groups of schizophrenics treated with electronarcosis,⁸³ found that these patients retained their recovery to a significant degree for at least two years, and that those patients who could be followed up after five years were still well. She particularly stressed the need to continue maintenance treatments (one or so a month) to prevent relapse during the period of return to normal living, as well as the need for psychotherapy during that period.

Holt, Landau, and Vernon⁴⁴ and Landau and Holt⁴⁸ introduced low voltage unidirectional currents with superimposed spikes of highly variable intensity into the field of electronarcosis. They used a Reiter machine specially constructed for this purpose (RC 47). The flexibility of this machine gives the operator a high degree of control of the cardiorespira-

tory as well as of the muscular response, thus practically obviating the necessity of having to interrupt treatment. The method they used is otherwise similar to that of Frostig,³⁴ Thompson,⁸¹ and Tietz⁸⁵ and their coworkers, in that a modified grand mal convulsion is followed by non-convulsive electric stimulation. Holt and his coworkers, however, named their treatment, "electric coma," a more appropriate term than electro-narcosis. An outstanding distinction of the work of Holt and his coworkers is their high standards of clinical controls. Only patients who had failed with one or more courses of standard electroshock treatment were chosen, thus eliminating the possibility of including patients who might have responded to any type of convulsive electric treatment. Their study is furthermore controlled by a parallel series of similar cases of failure after electroshock, which were treated with deep insulin coma treatments. The authors concluded that electric coma is superior to conventional electroshock in the treatment of schizophrenia, but inferior to insulin coma: 53 per cent of the electroshock failures were much improved after deep insulin coma treatment, while only 20 per cent were much improved after electric coma treatment, the classification of "much improved" being defined as consisting in disappearance of psychotic symptoms associated with good social adjustment, confirmed by reevaluation one year after discharge from the hospital. Patients who failed on one of the two coma therapies were subsequently treated with the other. Patients who failed with deep insulin coma treatment tended to fail also when subsequently treated with electric coma, and vice versa. Patients who were but slightly improved on one type of coma treatment occasionally obtained some significant benefit from the other.

I can confirm the authors' conclusion that electric coma is of distinct value in the treatment of schizophrenia from my own clinical experience. One patient who relapsed soon after I administered adequate electroshock as well as deep insulin coma treatment—34 electroshocks and 60 deep insulin comas were given—as well as after a subsequent lobotomy, obtained a sustained remission when I gave her a subsequent series of only 7 electric coma treatments. Another patient, who had failed on insulin and electroshock and who relapsed soon after lobotomy, obtained a good and sustained remission after 38 subsequent electric coma treatments of which 23 lasted a minimum of 7 and a maximum of 10 minutes. These cases certainly illustrate the precept that a therapist must never give up. Banay^{9,10} used electric coma in paranoid states and in aggressive psychopathic personalities, and reported favorable results.

NONCONVULSIVE ELECTRIC STIMULATION TREATMENTS

It appears that the first purely nonconvulsive electric treatment was given by no one less than Babinski,⁷ who presented a paper on the therapeutic effect of what he called "electric vertigo" before the Neurological Society of Paris on May 7, 1903; the paper, however, was not published until 1950. From the description it would seem that Babinski gave his patient two treatments, the first of which I would classify as nonconvulsive treatment of vasovagal type, the second as a myoclonic treatment without grand mal convulsion.

Berkwitz^{12, 13, 14} was the first to report on a large series of patients treated with nonconvulsive electric shocks. He used a simple apparatus

of his own construction consisting of an induction coil of the type used in automobile engines supplied from a 6-volt dry cell battery. Faradic high frequency type currents from this machine measuring 10 to 20 milliamperes in strength were administered from forehead to occiput. Current was allowed to flow for a total time of $7\frac{1}{2}$ seconds in an interrupted manner. After each $\frac{1}{2}$ second of flow of current, the current was interrupted for $\frac{1}{2}$ second. The total time of treatment, including these brief periods of interruption of the current, was 15 seconds. These treatments were administered daily over 10 to 20 days. Berkwitz stated that his currents were not powerful enough to produce unconsciousness. The patients were not even momentarily stunned, although momentary fear and excitement were produced. When stimulated the patients invariably shut their eyes tight and held their extremities rigid. The muscles of the face and neck contracted spasmodically with each shock, the face was flushed, the pupils were dilated, the pulse rate increased, the blood pressure was elevated, and salivation was increased. Respiration was not interfered with by the treatment. Because of the excitation and fear, it was necessary to quiet the patients following the treatment with intravenous pentothal sodium or sodium amytal. Berkwitz noticed that when, during subsequent administration of the intravenous barbiturate, respiration became irregular, stimulation with a few additional faradic shocks quickly revived the patients. Later Berkwitz¹³ administered the pentothal sodium simultaneously with the treatment. Berkwitz regarded his treatment as psychophysilogic, meaning that it had psychologic as well as physiologic effects. He reported rather uniformly good results in a wide variety of conditions, ranging through depression, hysteria, schizophrenia, and various organic psychoses, including those associated with alcoholism, drug intoxication, and syphilis.

Brief electroshocks, at nonconvulsive levels, can, of course, be administered through any of the standard electric shock machines. Androp⁶ administered such nonconvulsive treatments to a series of schizophrenic patients. He found the results of such subconvulsive treatment were almost as good as those obtained in a parallel series of patients treated with electroconvulsive therapy. However, he stated that nonconvulsive treatment failed to produce favorable results unless the current was strong enough to produce amnesia. This was not the case in Berkwitz's series.

Gottesfeld and his coworkers⁴⁰ reinvestigated the question of the therapeutic efficacy of brief nonconvulsive shocks in 1944, each subconvulsive treatment consisting of five applications given over a three-minute period. Such subconvulsive shocks were found to be of no therapeutic value whatsoever. Different electrode placements in no way modified this negative result. Kalinowsky and Hoch⁴⁶ likewise came to the conclusion that nonconvulsive shocks are worthless in psychotic patients and at times even tend to aggravate the patient's condition.

The first author to administer nonconvulsive treatment by means of direct currents of the type such as is used today was Delmas-Marsalet.^{25, 27} He observed and described the neurophysiologic phenomena of nonconvulsive stimulation of the brain with remarkable acuity. He differentiated three definite levels of response, depending upon current strength:

(1) *Minor seizure*—"absence" without apnea. This is accompanied by vasomotor reactions (first flushing, then paling of the skin) and modi-

fication of respiratory rhythm. When respiratory rhythm was irregular before treatment because of emotion or manic excitement, it became regular throughout the nonconvulsive treatment. Consciousness is usually abolished for about 6 or 7 seconds, seldom slightly longer. During this period of unconsciousness automatic movements may be noted, such as hands reaching for electrodes, touching the genitals, more complex movements such as buttoning and unbuttoning, seizing hands of the attendant, and so on.

Delmas-Marsalet regarded this "absence without apnea" as the result of diencephalic stimulation, although he contributed no evidence to prove this assumption. When slightly more intense currents were used, the second stage was reached, namely:

(2) "*Absence*" with apnea. Delmas-Marsalet observed that this apnea lasted up to 45 seconds. Connected with this is a loss of consciousness lasting about 3 minutes, then gradual return of consciousness. Flushing occurs in the initial stages of the treatment, later paling.

The third stage is reached when still higher currents are used, resulting in:

(3) "*Absence*" with muscular contractions, however, without grand mal convulsion.

Delmas-Marsalet stated that these nonconvulsive treatments which he termed "electrically induced absences" are especially beneficial in those depressions in which hypochondriasis is an important symptom. In these cases they are better than standard electroconvulsive treatments. He also regards nonconvulsive treatment as valuable in the manic phase of manic-depressive psychosis and for the manic symptoms of other periodic psychoses. In these conditions he finds this treatment superior to standard convulsive electroshock, because these treatments bring about a marked calming effect. In all other cases nonconvulsive treatment is inferior to convulsive electroshock. This is an important observation which I can fully confirm, especially the markedly calming effect of nonconvulsive treatment in manic psychoses. I assume that Delmas-Marsalet's term of "hypochondriac depression" may be synonymous with what I would call depression complicated by anxiety. In that case I can confirm Delmas-Marsalet's important observation in this respect also. He also found that nonconvulsive treatment has therapeutic value only when it results in loss of consciousness and that its therapeutic value is greater when it is associated with apnea than without.

Delmas-Marsalet introduced the practice of using nonconvulsive treatment at the beginning of an electric treatment series. If no improvement was noted after 6 such treatments he changed to standard electroconvulsive treatment. He also used nonconvulsive treatment whenever convulsive treatment failed or when there were contraindications to convulsive treatment.

Hirschfeld^{41, 42, 43} independently corroborated these findings in nearly all respects. He was, however, more specific than Delmas-Marsalet in noting the therapeutic effect of nonconvulsive treatment upon anxiety. As a practical measure in the administration of such nonconvulsive treatment, he modified Berkwitz's procedure in that he administered pentothal sodium prior to the electric treatment, thus administering the treatment under anesthesia except in certain cases of schizophrenic psychosis in which he

preferred to give the treatment without anesthetic. He also followed Delmas-Marsalet in interpreting the vegetative phenomena produced as due to stimulation of the diencephalon. However, like Delmas-Marsalet he failed to establish convincing proof of this assumption.

Most of the investigators of electronarcosis, electric coma, and purely nonconvulsive stimulation treatments, however, have used the nonconvulsive stimulation as an extension of, as an alternative to, and as a supposed improvement over convulsive shock therapy, although some, particularly Delmas-Marsalet,²⁷ Wilcox,⁹⁵ Goldman,³⁹ Hirschfeld,^{41, 42} and Banay¹⁰ had noted that it was different in that it was particularly effective in certain groups of patients and conditions.

Countershock. In my work with nonconvulsive treatment (Alexander⁴), I decided to find out more about the precise neurophysiologic and psychophysiological nature of this difference. Early in my study of the literature and reports of the work on electronarcosis—although this was not stated specifically by any of the authors—I felt that the nonconvulsive part of the so-called electronarcosis treatment did not actually merely extend the therapeutic effect of the convulsive part of the treatment, that it was not only different from convulsive treatment, but that it was actually antagonistic to it, in that it modified, altered, and to some extent reversed its effect. If this supposition proved true, I felt that it would provide an opportunity for quantitative study of the effects of electric treatment—convulsive or otherwise. If a mutually cancelling or otherwise quantitatively interfering effect could be proven, it would provide opportunity for a scientific study of the psychophysiological phenomena which probably constitute the basic principles of electric treatment techniques. The likelihood that such could be the case was suggested by my knowledge of the studies on “countershock,” discovered as early as 1775 by Abildgaard,¹ later proposed as a practical method of resuscitation by Campbell and Hill,¹⁸ and studied in detail by Hooker, Kouwenhoven, and Langworthy,⁴⁵ and Ferris, King, Spence, and Williams.²⁸ I had become familiar with these studies in my earlier work on accidental electrical injuries (Alexander²).

The principle of “countershock,” new to the electric treatment of mental disease, was put to the test in the fall of 1948 when I treated a woman suffering from a severe depression, who after each convulsive electric shock developed a most alarming and prolonged state of apnea and laryngeal spasm, requiring each time maximum prolonged exertion of resuscitating efforts including oxygen, artificial respiration, and various stimulants. Several months before this incident I had had to terminate prematurely the treatment of a similar patient for the same reason. The alarming reaction had become worse with each subsequent treatment so that after the tenth treatment she almost died. When I subsequently terminated her treatment series and recommended lobotomy, her family refused to carry out the recommendation and she remained unimproved. Since I did not want to give up again with this second case and most importantly, since in the meantime my ideas on the possibility of countershock had crystallized, I decided to make her the first case for the test of the countershock procedure. As soon as the convulsion elicited by the electroshock was completed and while the patient lay there in her usual state of deep apnea and laryngeal stridor, I applied a countershock of low voltage with the aid of the Reiter CW46 machine. A low intensity shock of only 1 to 1.5 milli-

amperes was chosen in order not to set off another convulsion. Immediately upon application of the countershock, the patient started breathing regularly. Some remnant of laryngeal spasm was still recognizable for a short time by the peculiar forced nature of her respirations, but this gradually subsided. Her color changed from the blue-gray of deep cyanosis to a healthy pink. This technique was repeated not only in subsequent treatments in her case but also in other patients with lesser states of apnea and it was found that the procedure worked.

It soon became apparent that not only did the nonconvulsive countershock relieve the respiratory and circulatory side effects of the convulsive electric shock treatment, but it rather remarkably also changed the state of the patients' post-treatment activity. Those patients who had previously been content to sit about inert for the remainder of the day after treatment started to play cards and take walks outdoors. The rather pronounced memory defects cleared up promptly. Postshock anxiety, when brief countershock through frontal leads was given, became sometimes more pronounced, but when the time of the countershock was lengthened or countershock given through temporoparietal leads, postshock anxiety was likewise greatly relieved. Soon this new method of shock-countershock treatment encouraged me to treat patients whom I previously would not have dared to treat by electroshock, such as a woman with a depression following ligation of a large intracranial carotid aneurysm and a depressed woman with a severe degenerative disorder of the nervous system. The new method was presented at a meeting of the Boston Society of Psychiatry and Neurology on February 17, 1949,⁵ and subsequently at a round-table discussion of the American Psychiatric Association on May 23, 1949.³

Until the preliminary publication of Hirschfeld's work⁴¹ in August, 1949, I had given most of my nonconvulsive treatments during the period of unconsciousness following convulsive electric shock, since on two occasions when volunteer patients allowed me to give them nonconvulsive treatments not preceded by convulsive electroshock the experience was so unpleasant for the patients that the attempts were discontinued. In the summer of 1949 I learned of Hirschfeld's nonconvulsive method with pentothal sodium. I subsequently used his technique as well as my own, but found Hirschfeld's method had the same effect in modifying the side effects of previously administered electroshock therapy as my own immediate application of nonconvulsive current: namely, it restored apparent memory defect caused by convulsive electroshock; it caused greater activation and it relieved postshock anxiety. On the other hand, I soon found that when such nonconvulsive treatment was given to depressed patients prior to electroshock, it enhanced the depression, obviously by further breaking down of the defenses. It was also found that major psychoses were rarely benefited by nonconvulsive treatment alone; however, when used in combination with and particularly in judicious alternation with convulsive therapy, it could be used to steer the patients through the Scylla of depression on the one hand and the Charybdis of anxiety on the other. It also could be used to tone down anxiety prior to the beginning of the convulsive treatment series and to restore memory defect as well as alleviate anxiety at the end of the convulsive treatment series. Its use as an adjunct to psychotherapy in anxiety neuroses and residual neurotic states following

recovery from schizophrenic psychoses appeared likewise promising in a number of cases, although in this respect it was distinctly inferior to insulin, and it proved sometimes inferior to abreaction by ether or sedation and relaxation by insulin subcoma.

PSYCHOSURGERY

The most important method of physical treatment to antedate (by two years) the introduction of electroshock treatment was lobotomy, a surgical procedure consisting essentially of a severance of the connection between thalamus and frontal lobe. This method was first used effectively by Burckhardt¹⁷ in 1891, but subsequently fell into oblivion. Egas Moniz⁶⁴ rediscovered the method when he was stimulated by Fulton's and Jacobsen's³⁵ experiments on monkeys which demonstrated increased docility and acceptance of frustration following lobotomy, and encouraged by Brickner's^{15, 16} careful study of a patient who had undergone bilateral frontal lobectomy for the sake of removal of a brain tumor, resulting in a carefree, careless, unworried, uninhibited state, without severe intellectual or significant psychobiologic deficit. Frontal lobotomy for mental disease has proved a very effective procedure in those patients in whose illness hostility and tension play a significant part, including hostility to the self. Moniz's method might have shared the fate of Burckhardt's work had it not been for the vigorous perseverance of Walter Freeman and J. W. Watts,^{29, 30} who introduced the method in America and persevered in its development in spite of academic objections.

That lobotomy affects the reverberations between the thalamus and the frontal lobes has been proven by the electroencephalographic work of Margaret Lennox and J. Coolidge,⁵¹ whose findings have been confirmed in our own clinical experience at Boston State Hospital. These authors found that the barbiturate-induced sleep-spindles that originate in the interbrain are no longer transmitted to the frontal lobes following lobotomy, nor, we found, are they any longer transmitted in their former intensity to other parts of the brain.

The side effects of lobotomy were prominent in the early phase of its development when frontal lobe destruction was extensive and relatively uncontrolled. Subsequent improvements in technique and increased insight into functional relationships have led to considerable decrease in the undesirable side effects. Visual control over the originally blind operation, for instance, was introduced by Lyerly^{57, 58} in 1947 and by Poppen⁶⁷ in 1948, selective undercutting of specific areas by Scoville⁷⁶ in 1948, selective excision of cortical areas by Penfield and his coworkers in 1948,⁶⁶ and by Mettler and his associates in 1949.²¹ Hamlin recently combined selective undercutting with minimal operations on the central white matter of the frontal lobes, based on his anatomic studies with Yakovlev and Sweet.⁹⁷ Recently Ward^{91, 92} and Livingston⁵⁶ have worked on undercutting the anterior cingulate gyrus (area 24) only.

From the point of view of scientific method and procedure the most important method of comparative cortical ablation of the frontal lobes appears to be in the development of topectomy, a rather thorough study of which has been made by the Columbia-Greystone Associates (Mettler and his coworkers) in their book, *Selective Partial Ablation of the Frontal Cortex*.²¹

This is a well documented report of a thorough study of 24 cases of mental disease in which selective ablations of specific areas of the frontal cortex were carried out. This new selective operation is called topectomy by the authors. It consists of removal of a specific area of gray matter, in contrast to the standard form of frontal lobotomy which consists of incisions into the anatomically less well differentiated white matter (leukotomy), or to gyrectomy (Penfield,⁶⁶), which consists of the removal of specific gyri.

In addition to the 24 topectomized patients, 24 control patients were studied with equal thoroughness; in 8 of these the standard leukotomy was performed.

The authors embarked upon their new surgical approach in an effort to learn whether small and specific ablations of well circumscribed areas of the frontal cortex—and if so, which ones—might bring about the same beneficial effects that leukotomy (the standard form of frontal lobotomy) does but without the undesirable and unavoidable side effects of the extensive standard operation. Prior to this work, it was an open question whether or not the extent of the operation per se had something to do with its effects (a not inconceivable possibility in view of Lashley's work on animals) or whether interference with certain specific cortical systems was responsible for the desirable results. If the latter were the case, then certainly it should be possible to minimize the undesirable side effects by narrowing the area operated upon. It was the wise decision of the authors to remove the much better defined cortical areas rather than the interwoven and anatomically far less differentiated fiber tracts of the white matter.

Although the series of cases at the authors' disposal was small, they nevertheless have produced convincing evidence that the beneficial effects of frontal lobe operation is not based on the total amount of frontal lobe tissue removed. No correlation was found between the clinical result and the amount and weight of gray brain tissue removed. Desirable clinical results were found to be based on the removal of, or significant encroachment on, areas 9 and 10 as well as the small adjacent area 46. In the 13 of the 24 topectomized patients in whom interruption of psychotic behavior resulted, one of these three areas had been removed wholly or in part. Of those patients in whom one of these three areas had not been encroached upon, none recovered.

The important findings that the authors have obtained with their method of localized cortical ablations will undoubtedly be a valuable incentive to efforts aimed at improving the clinical results of lobotomy by more limited and circumscribed incisions into the white matter; and these efforts will be significantly helped by the careful study of cortical ablations performed by the authors.

Other new and potentially valuable surgical procedures are being developed and are of course in need of further study and research. A few of these are Gibbs' and Bailey's work^{8,36} on removal of anterior parts of the temporal lobe to abolish uncontrolled aggressive fugue states in severe psychomotor epilepsy, and a recent attempt reported by Williams and Freeman⁹⁶ to suppress auditory hallucinations by removal of the amygdaloid nucleus.

GENERAL PRINCIPLES OF THE EFFECTS OF PHYSICAL
METHODS OF TREATMENT

The manner in which all these treatments work—the why of their effectiveness—is still obscure, although most workers in the field agree that the primary change is one of mood. From perusal of the literature and from my own clinical experience, I have come to the conclusion that the primary change in mood is particularly significant in that it seems to exert a profound influence on the relative level and balance of psychologic dynamics; different constellations of the dynamic balance of the personality seem to emerge with varying changes in mood and energy level. Rennie⁷⁰ has expressed this same idea in regard to the effects of insulin coma. He feels that the basic anxieties are most strongly influenced by such treatment and that this in turn seems to effect the lowering or disappearance of specific anxieties. He comments: "It seems possible that anxiety, like other affects, may lie behind delusional development only to be ultimately incorporated into the delusional system so that its role is no longer clearly seen. Anxiety tends to keep dynamic factors alive and relief of anxiety permits them to subside,"—an observation in keeping with current psychodynamic theory concerning the association between anxiety and defensive operations.

In my own studies I find that defensive operations seem to be affected in specific ways by various methods of physical treatment. It would seem probable that the changes in defensive operations thus brought about are only indirectly results of the physiologic changes induced by physical treatment, that these physiologic changes act rather as a substratum, an initiating event that sets into motion a series of changes and shifting balances that eventually enable the personality to reorganize its approach, and regroup its defenses. (This will be further elaborated later.)

In general, the effect of electroshock treatment appears to be such that the person is somehow enabled to strengthen certain defenses and to replace poor or non-efficient ones with better ones. Following successful electroshock treatment the patient may be able to attack his problems and conflicts with a different system or balance of defenses, for instance, by effective reaction formation resulting in altered self-perception in which he now sees himself as good. Sometimes he seems able to enlist an effective defensive method in isolation, or even in denial—or perhaps more accurately, in increased tolerance for frustration, pain, and anxiety so that he can now afford to ignore or put into different perspective previously very provoking matters. This last reaction is particularly striking in lobotomy in which the cure of pain, for instance, is essentially based on a method of denial resulting in the patient living as if the pain did not exist, although direct questioning may draw the admission that pain is still there. This may in part be increased tolerance, rather than denial, in pain cases as well as in patients operated on for mental disease—that is, an ability to tolerate or disregard, whereas denial connotes a primitive, purely unconscious defensive operation. I have, however, obtained convincing evidence of the effective use of denial as a defense after lobotomy, not only in my own patients, but also in published observations of statements, attitudes and actions after lobotomy in the literature. This will be discussed in more detail in later chapters.

Nonconvulsive electric treatment, on the other hand, seems to enable the patient to lower his defenses and enables him to come to grips with his real problem and reorganize himself on a more realistic basis. In view of the seemingly specific action of nonconvulsive treatment on certain types of anxiety, this psychologic change may result from the fact that the crippling anxiety that previously served to keep the patient welded rigidly to his defensive maneuvers has been sufficiently allayed so that he may now relax them and survey his situation in a more realistic manner.

In insulin treatment, phases of both these processes are observable—that is, strengthening of certain defenses and lowering of others. A young schizophrenic woman who prior to receiving insulin coma treatment had been in psychoanalysis for two years with an excellent analyst was seen again by him half way through her treatment, after she had undergone 30 insulin comas, and at a time when she showed considerable improvement, particularly in terms of lowered tension. The analyst noted for me his impressions of the change that had taken place in this girl, and there are two items of particular interest, one of which indicates a lowering of her defenses. This observation was occasioned by her reaction to a particularly deep coma: “When she inquired as to why that morning had been different and you remarked that she had gone deeper into her coma, she seemed to me to pick it up in her old covert anxious way. The old, or rather, former drive to seek out cruel bloody disaster seemed on the hunt. You have a word, ‘thanatolatry,’ which struck me as an apt characterization of this drive in her. Was I seeing a hint that it still existed? The surface indication for it in analysis was always very fleeting and hidden.”

The second item may again indicate less reliance on former more rigid modes of defense and at the same time the emergence of more healthy defenses in terms of affective bonds and the disappearance of her former tendency to project: “In describing her emergence from coma she gave three stages: (1) a dim awareness ‘I am me’; (2) an awareness ‘There is more than me around here’; (3) ‘These others are the doctor and nurse’; then full awareness, and (4) a return of the ability to move. What is missing in this account is the feeling, ‘These others are my enemies doing something awful to me,’ which I take it she would once have had. What was a hypochondriacal self-awareness has given way to a more objective, simple self-awareness.”

Projection as a means of defense is frequently the first one to be replaced by a normal defensive operation, namely, identification, which is a prerequisite for the acceptance of all psychotherapy as well as of all teaching, and of cultural integration in general.

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VIII

THE ELECTRICAL PROPERTIES OF CURRENTS USED FOR TREATMENT

TYPES OF CURRENTS USED

The physician using electric treatment must understand not only the physiologic events taking place, but also the parameters of electrical stimulation that he employs. Thorough understanding of both these factors is necessary for proper mastery of the techniques available at present, and is particularly prerequisite for development and fruitful utilization of further advances in technique. The following characteristics of therapeutic currents are of particular significance: (1) basic type of current and wave shape; (2) duration of the pulses used; (3) milliamperage used, both in regard to the average current and its peak; (4) the total duration of treatment as well as the length of the individual volleys employed; (5) the mode of current onset, i.e., whether or not the onset is instantaneous or whether there is a slow rise. For the latter, the term "glissando" has been introduced by Tietz and her coworkers¹⁵; (6) electrode size and placement; (7) resistance or impedance of the subject and the apparatus; (8) the rheobase of the nervous system of the subject (Wilcox and Adler¹⁸); (9) voltage of the stimulation.

Electric currents may be divided into three basic types: (1) direct current (d.c.); (2) alternating current (a.c.); (3) pulsating (unidirectional) current (u.c.) (Fig. 3). For electric treatments in mental disease, alternating currents and pulsating currents are used. The wave form of the alternating current used is that of the sine wave (Fig. 4). Pulsating currents consist of the following wave forms: (1) Rectified sine waves, which include half-wave rectification, full wave rectification, and filtered rectified currents. (2) Rectangular waves, also called square waves. These are employed in brief stimulus therapy (Liberson¹³). (3) Exponential or sawtooth waves, such as those used in the thyatron stimulator. (4) Amplitude modulation of the above basic wave forms, such as used in the Reiter machines (Fig. 4).

Convulsive stimulation with alternating currents has a very low impedance, the current in the path being stimulated being proportional to the electrode resistance which generally measures about 360 ohms. Thus, a 60-cycle current of 120 volts (root mean square) yields an average current of 330 milliamperes, measuring 924 milliamperes from peak to peak (Fig. 5).

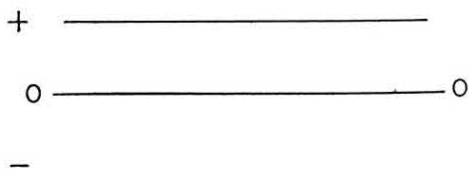
Most alternating current machines operate strictly on a line voltage and

frequency, the current being modified by an isolation transformer. The glissando attachment, operation of which is optional, uses a motor-driven potentiometer to obtain a slow rise (Fig. 5, B) of current intensity. The frequency, however, remains entirely fixed.

Liberson's brief stimulus therapy machine, manufactured by Offner, allows for frequency modulation as well as for control of the duration of the individual pulses (Fig. 6).

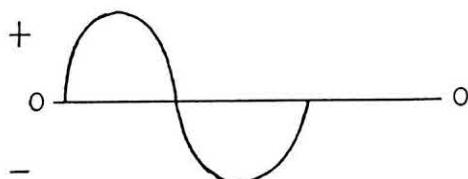
DIRECT CURRENT (d.c.)

An electric current having a constant flow in only one direction and also having constant polarity. (As obtained from dry cell or storage battery.)



ALTERNATING CURRENT (a.c.)

An electric current which periodically reverses its direction of flow. In addition, the amplitude continually varies. (As found in common house current usually 60 c.p.s.)



PULSATING UNIDIRECTIONAL CURRENT (u.c.)

Composed of an alternating current superimposed on a steady direct current. (Brain waves are probably of this type.)

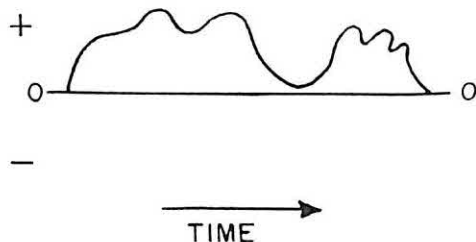


Fig. 3. Current nomenclature (basic types of current). (From Electroshock Research Association, Nomenclature Committee⁸.)

Reiter's machines employ automatic amplitude modulation of unidirectional pulsating currents, while their basic frequency remains fixed (28 per second) (Figs. 7 and 9). With the CW 46 stimulator the amplitude modulation is set at 4 per second (Fig. 7), while with the CW 47 and RC 47 stimulators, controls are provided which enable the operator to vary the amplitude modulation from 3 to 6 per second (Fig. 9).

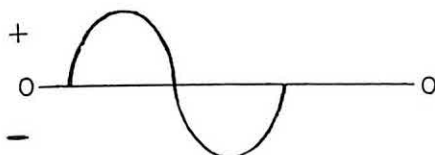
The individual pulse duration is relatively longer for the currents most suitable for convulsive stimulation (5 to 8 milliseconds, Fig. 8); relatively shorter for the currents most suitable for nonconvulsive stimulation (3 to 4.2 milliseconds, Figs. 10, B, C, D).

The internal impedance of the Reiter stimulators is higher and stimulating current is less dependent upon the electrode resistance than in the standard alternating current machines. Because of this, the consideration

of voltage output of these stimulators is not so important as the consideration of the measurement of the current itself. Measured with 1000 ohm

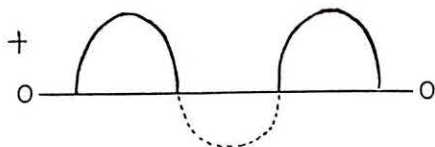
A. Alternating Current

SINE WAVE (Commonly used in Cerletti-Bini type EST at frequencies of line current, i.e., usually 60 c.p.s.).



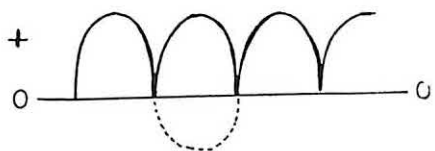
B. Pulsating Currents (unidirectional)

1. RECTIFIED SINE WAVE (Basic form for deriving so-called "unidirectional currents").

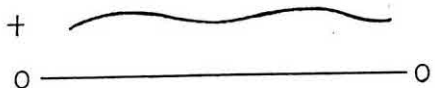


a. Half-wave rectification.

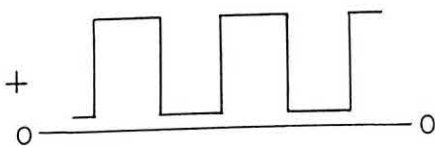
b. Full wave rectification.



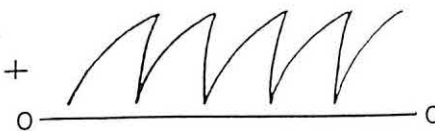
c. Filtered rectified current.



2. RECTANGULAR WAVES (Square waves used in BST Liberson-Offner stimulator).



3. EXPONENTIAL OR SAW-TOOTH WAVE (used in thyatron stimulator).



4. AMPLITUDE MODULATION OF THE ABOVE BASIC WAVE FORMS (used in Reiter stimulators).

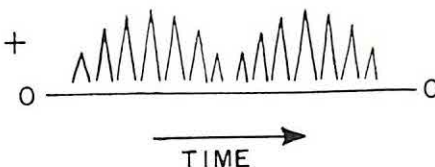


Fig. 4. Wave form nomenclature. (Modified from Electroshock Research Association, Nomenclature Committee⁸.)

resistance in patient circuit, the possible peak voltage was found to be 450 volts.

Currents suitable for convulsive electroshock treatment obtainable from the Reiter CW 46 stimulator and from the convulsive range (No. 3) of the CW 47 stimulator are composed of 28 pulses per second, with the amplitude modulated at four per second (Fig. 7). Each individual 28 per

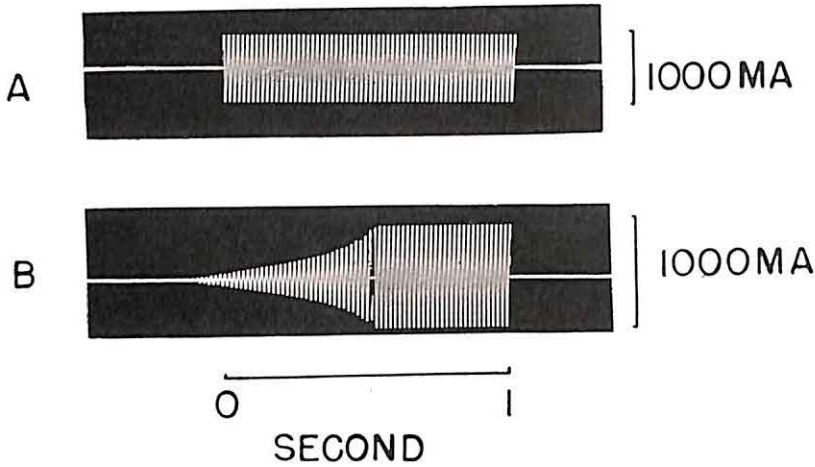


Fig. 5. A. Alternating current for convulsive treatment. Instantaneous onset. Sinusoidal wave form; 120 volts. Duration of flow of current, 1 second. Average current, 330 milliamperes, peak-to-peak current 924 milliamperes.

B. Alternating current, slow rise ("glissando"). Duration of slow rise, 0.6 second. Duration of flow of full strength of current, 0.5 second. 120 volts. Average current at full strength, 330 milliamperes; peak-to-peak current at full strength, 924 milliamperes.

Medcraft machine. Oscillograph pictures. One second equals 33 mm. This oscillograph tracing as well as the oscillograph tracings reproduced in Figs. 7, 8, 9, and 10 was made, photographed, and interpreted by Mr. Albert Grass of the Grass Instrument Company, Quincy, Mass.

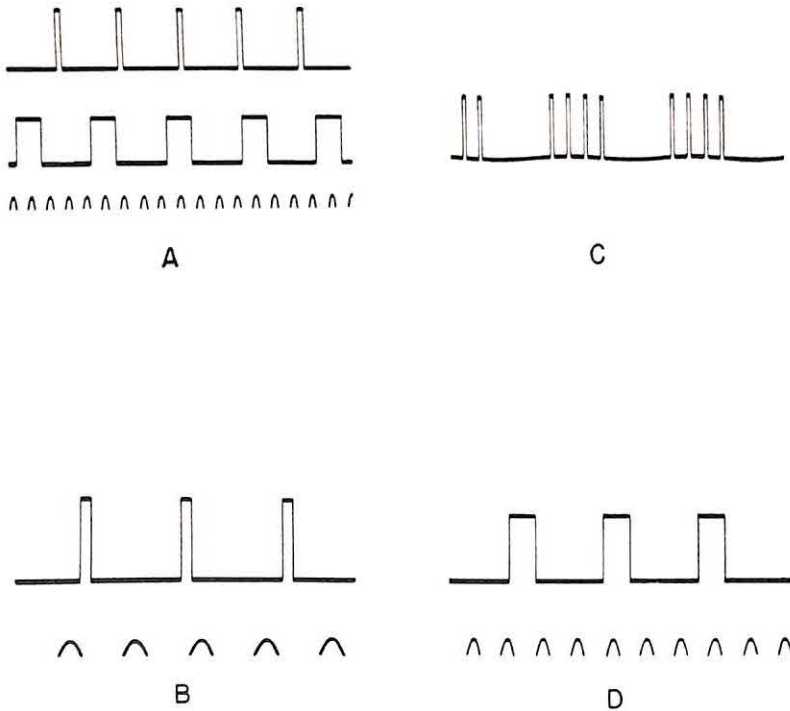


Fig. 6. Types of current produced by "Brief Stimulus Therapy" machine (courtesy of Drs. W. T. Liberson and F. Offner).

A. Upper line. 0.3 millisecond rectangular spikes with interval of 3 milliseconds (about 300 per second). Middle line. 1.5 milliseconds spikes with the same interval. Lower line. Time: 1000 cycles per second.

B. 2 milliseconds spikes with interval of 15 milliseconds. Time: 1000 cycles per second.

C. Groups of spikes with interval between groups of 20 per second.

D. 1 millisecond spikes with interval of 2 milliseconds. Time: 1000 cycles per second.

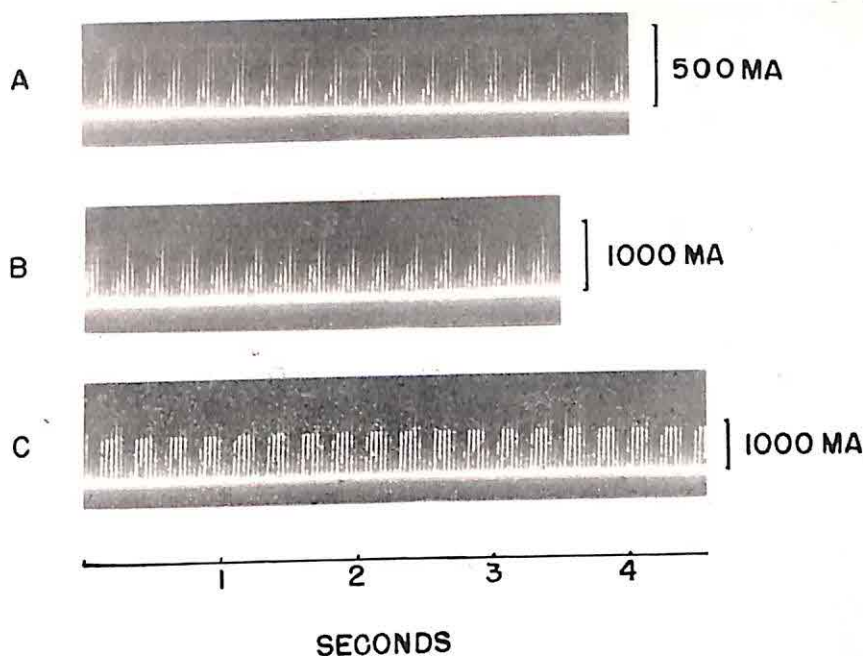


Fig. 7. Amplitude modulated unidirectional currents, convulsive range, obtained from Reiter CW 46 machine, Number 3 position. Oscillograph pictures. One second equals 17 millimeters.

- A. Average current of 5 milliamperes with spikes up to 370 milliamperes.
- B. Average current of 15 milliamperes with spikes up to 925 milliamperes.
- C. Average current of 20 milliamperes with spikes up to 1670 milliamperes.

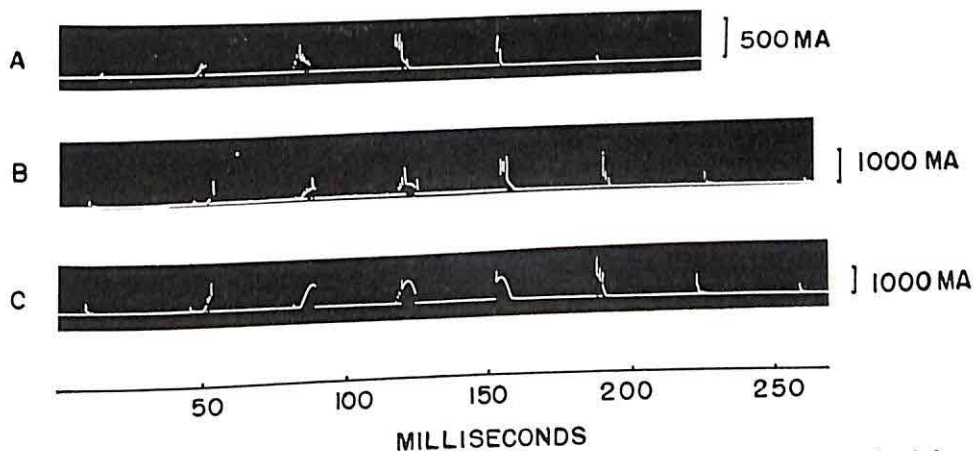


Fig. 8. Amplitude modulated unidirectional currents, convulsive range, obtained from Reiter CW 46 machine, Number 3 position. Oscillograph pictures. One millimeter equals 2.8 milliseconds.

- A. Average current of 5 milliamperes with spikes up to 370 milliamperes.
- B. Average current of 15 milliamperes with spikes up to 925 milliamperes.
- C. Average current of 20 milliamperes with spikes up to 1670 milliamperes.

second pulse is random in its shape and of 5 to 8 milliseconds duration, separated by intervals of 25 to 30 milliseconds duration (Fig. 8). Each of these pulses in turn contains sharp spikes of less than 1 millisecond duration. The variation of these spikes as well as the variation in the shape of the pulses is in part derived from sparking of the contacts. This is in

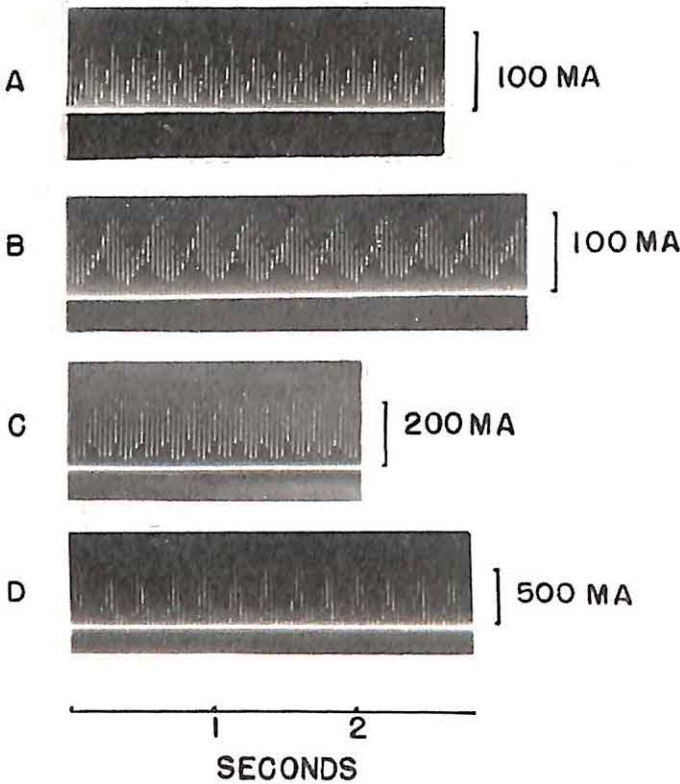


Fig. 9. Amplitude modulated unidirectional currents, nonconvulsive range. Oscillograph pictures. One second equals 17 mm.

A. Reiter's CW 47 machine, Number 1 position, fast modulation. Average current of 2.5 milliamperes with spikes up to 98 milliamperes.

B. Reiter's CW 47 machine, Number 1 position, slow modulation. Average current of 2.5 milliamperes with spikes up to 98 milliamperes.

C. Reiter's CW 47 machine, Number 1 position, fast modulation. Average current of 10 milliamperes with spikes up to 252 milliamperes.

D. Reiter's RC 47 machine (electric coma machine), Number 3 position, Medium fast modulation. Average current of 4.5 milliamperes with spikes up to 623 milliamperes.

contrast to the electronic type of stimulator in which uniform repetition of pulse shape and duration predominates.

As the average current is raised from $\frac{1}{2}$ milliamperes to 15 milliamperes, the ratio of the peak value of the current to the average value decreases (Table 1). This is primarily due to the fact that as current is increased, the individual pulse envelope progresses to contain relatively more energy than the corresponding increase in the peak value of the spikes (compare Figs. 10 A, 8 A and 8 B). As the current is further increased to 20 milliamperes of average current, however, the pulse envelope not only contains more energy, but also rises greatly in amplitude (Fig. 8 C), thus en-

hancing the total amplitude composed of the pulse envelope and the superimposed spikes. Correspondingly, the ratio of the peak value of the current to its average value rises again at this level (Table 1).

Currents suitable for nonconvulsive stimulation obtained from Reiter machines likewise show a pulse rate of 28 per second, but the modulation frequency can be varied. When maximally fast modulation is utilized, this

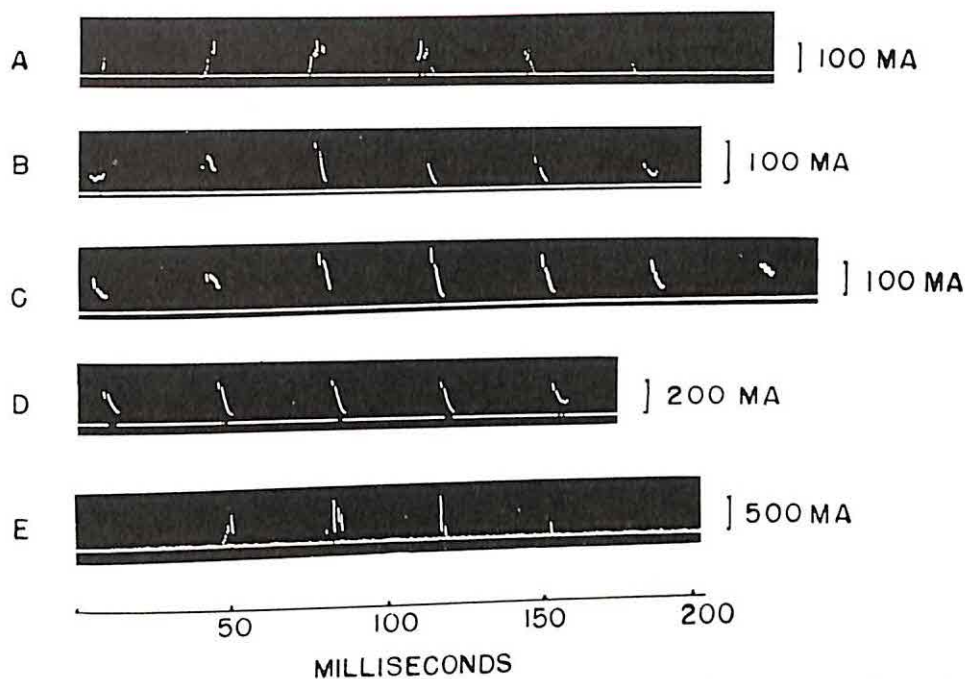


Fig. 10. Amplitude modulated unidirectional currents, nonconvulsive range. Oscillograph pictures. One millimeter equals 2.6 milliseconds.

A. Reiter's CW 46 machine, Number 1 position. Average current of 1 milliamperes with spikes up to 137 milliamperes.

B. Reiter's CW 47 machine, Number 1 position. Average current of 2.5 milliamperes with spikes up to 98 milliamperes.

C. Reiter's CW 47 machine, Number 1 position. Average current of 5 milliamperes with spikes up to 160 milliamperes.

D. Reiter's CW 47 machine, Number 1 position. Average current of 10 milliamperes with spikes up to 252 milliamperes.

E. Reiter's RC 47 machine (electric coma machine), Number 3 position. Average current of 4.5 milliamperes with spikes up to 623 milliamperes.

modulation frequency is increased to 6 per second (Figs. 9 A and 9 C). When maximally slow modulation is utilized, this frequency is 3 per second (Fig. 9 B). With electric coma currents, the modulation usually employed for coma is 5 per second (Fig. 9 D). As to the individual pulses in the nonconvulsive range of currents obtainable from the CW 47 machine, they are of shorter duration, measuring from only 3 to 4.2 milliseconds each, separated by intervals measuring 30 to 35 milliseconds. Their shape is more uniform, being essentially that of an exponential condenser discharge with relatively few spikes within the pulse envelope (Figs. 10 B, C, and D). The nonconvulsive currents obtainable from Reiter's CW 46 machine are slightly longer in pulse duration, namely up to 5.8 millisec-

onds, and the spikes composing each pulse are more numerous (Fig. 10 A); electric coma currents are similar in this respect (Fig. 10 E).

Table 1. The Quantitative Relationship of Average Current Values to Peak-to-Peak Current Values

TYPE OF CURRENT AND MACHINE	AVERAGE CURRENT	PEAK-TO-PEAK CURRENT	RATIO OF PEAK-TO-PEAK CURRENT VALUE TO AVERAGE CURRENT VALUE
			×
a.c., Medcraft machine	330	924	2.8
	500	1400	2.8
u.c., Reiter's CW 46 machine	$1\frac{1}{2}$	70	140
	1	137	137
	2	200	100
	3	270	90
	5	370	74
	10	650	65
	15	925	62
	20	1670	84
u.c., Reiter's CW 47 machine (nonconvulsive range)	$2\frac{1}{2}$	98	39
	5	160	32
	10	252	25
u.c., Reiter's RC 47 machine (electric coma machine)	$4\frac{1}{2}$	623	139

THE PASSAGE OF ELECTRIC CURRENTS THROUGH THE INTACT BODY

The problem of the mode of passage of electric current through the living body has been a rather vexing one. A good many of the older authors believed that electric current traveled through the body along blood vessels and nerves. Weeks and I¹⁶ disproved that notion by establishing the fact that electric current of the frequency, voltage, and amperage prevailing in most household and industrial accidents and employed in therapeutic electroshock passes through the living body as though it were passing through a structureless gel, always choosing the shortest path from contact to contact without deflection by anatomic landmarks. The outer boundaries of this current path are usually not straight lines, but elliptic or sometimes paraboloid curves (see Weeks and Alexander,¹⁶ Figures 4, 5, and 6). The density of the current diminishes along the margins of the curved outer boundaries of the path. This diminution in density is greatest along the crest of the curve of the margin of the path. The technique which we used to establish these facts employed the insertion of an insulated small clamp transformer. We were, of course, fully aware of the fact that the various tissue substrates, when examined after removal and drying, vary greatly in their electrical resistance. In the living body, however, these differences appeared to be practically nonexistent, obviously because of the presence of the all-pervading tissue fluids and the circulating blood. The vascular bed pervades even living bone as completely as most other tissues of the body.

Our findings concerning the uniformity in the penetration of living body tissues by electric current are of course true only for currents within

the range of voltage, amperage, and frequency as specified above. It is known that very weak currents as well as very high frequency currents (Tesla currents) tend to be deflected along body surfaces and do not penetrate at all. This is due to the fact that the resistance of the body, including the skin and the various body tissues, increases greatly in an inverse order to voltage and amperage, as well as above a critical frequency, such as in the range of the high frequency currents of the Tesla type. Such a marked increase in resistance will also, of course, greatly magnify the existing differences in resistance as well. Within the range of voltage, amperage, and frequency with which we are dealing in electroshock treatment, however, these differences are negligible and all of the body that is enclosed within the skin will act comparable to a structureless gel. Such a gel, of course, still has greater resistance than either a metal conductor or a salt solution. This must be kept in mind in order to understand an apparent exception to our rule which Hayes¹⁰ recently brought up as an argument against the validity of its principle.

In order not to be misled by such apparent exceptions it is necessary to keep in mind the general principle of electrodynamics which is readily understandable in the light of its practical identity with the principle of hydrodynamics. In the same way that water will flow down along any altitude-gradient, so will electricity flow down any potential-gradient. The directness of the flow increases with the steepness of the gradient. Water will flow down straight over obstacles such as rocks when the altitude gradient is steep, but will flow around the rocks when the angle of the gradient is only slight. In the same manner, electricity will flow right across areas of relatively greater resistance when the potential-gradient is marked, while it will flow around the resistance-obstacle if the potential-gradient is slight. In other words, water as well as electricity will always flow down along the path of least resistance; but the intensity of the gradient will determine the relative obstacle value of the resistance encountered. Given a sufficient gradient of altitude or potential respectively, water or electricity will always flow in a more or less straight line over and across obstacles unless a great deal of inducement is offered in the form of an alternative course offering much less resistance, although it may be longer. The course of running water may be deflected by a pipe going a circuitous route, provided its core is free from obstacles. Electricity may flow along a shunt path offering less resistance than that offered by body tissue, such as in the case of a shunt path made of metal or saline solution. It is important to realize, however, that if the skin contact is good, even saline solution will not deflect much current from a straight path through the body. This fact was exemplified by a case of fatal electrocution through a urine-soaked heating pad (Alexander^{1, 2}). The conduction facilities through the animal body are indeed so good that cattle may be electrocuted by spanning current through the legs from a rain-soaked meadow, provided that there is insulating rock beneath the ground (Alexander³). Nevertheless, metal conductors will, of course, deflect current from its normally practically straight path through the brain because metal is a far better conductor than brain tissue.

It is obvious that the metal probes which Hayes¹⁰ employed to measure the voltage gradient within the brain deflected current in occipital direc-

tion in a "lightning rod" manner. I am certain that he would have found no appreciable current flowing through the occipital lobes if he had used insulated transformers. Another source of artefact in Hayes' study were the occipital trephine holes which had been drilled in the skull to admit the probes. These were obviously responsible for the otherwise paradoxical fact that almost as much current passed through the occipital areas as passed through the frontal areas from the frontally placed leads, while the intervening parietal and temporal areas of the brain passed only much smaller amounts of current. The availability of a shunt path by way of spinal fluid contact through the trephine holes was sufficient inducement

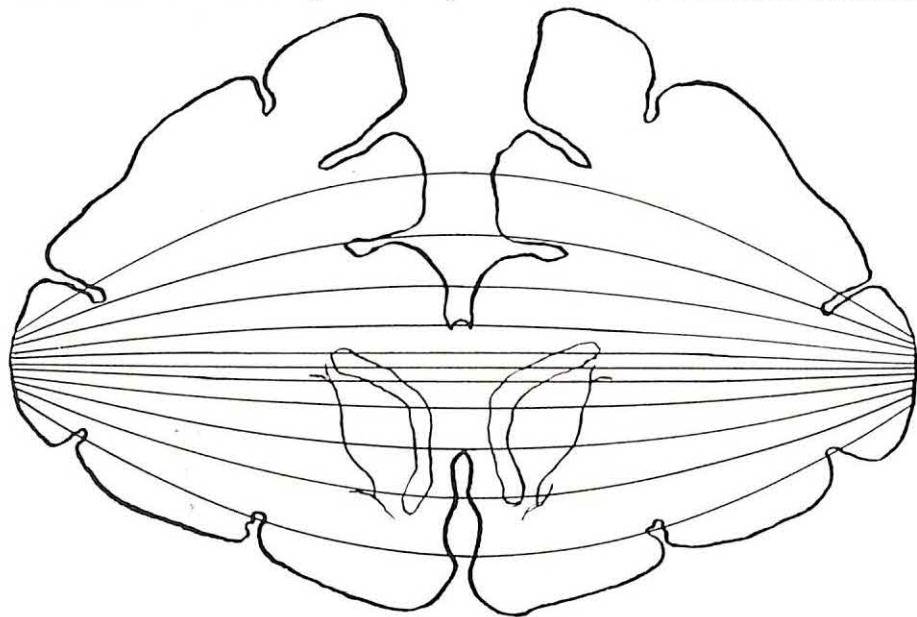


Fig. 11. The path of electroshock current applied through bifrontal leads across the brain, based on the study of the intracerebral vascular reaction as an indicator of the path of the current (after Alexander and Löwenbach⁴).

to deflect current through the occipital regions of the brain. Although this path is longer, it offers obviously less resistance because spinal fluid is a better conductor than any solid body tissues. Furthermore, in view of the small size of the head of the spider monkey, the resulting increment in length of the path is hardly very great. While this observation should be considered when administering electroshock to patients with trephine openings in the skull, we must conclude that for the intact skull the path of the current remains essentially that outlined by our previous studies (Weeks and Alexander¹⁶).

I repeated these studies in collaboration with Löwenbach,⁴ using a different method in outlining the path of the current through the skull and the brain. We utilized the vascular reaction within the brain which can be emblazoned by currents of sufficient strength, that is, by currents significantly above the therapeutic level but still falling into the same general low voltage category of current (2000 milliamperes flowing for 7 to 10 minutes). With this method we established that the current flowed along an essentially straight path from one electrode to the other across the

frontal lobes (Fig. 11). No biologically appreciable amount of current went through the parietal or occipital lobes, since not the slightest vascular reaction occurred in those areas. The vascular reaction was least intensive along the crest of the curve of the margin of the path as well as within the median part of the core of the path. We must assume, therefore, that there is also a slight spread and diminution of density of the current within those median parts of the central core of the path which correspond to the crest of the elliptic curve of the marginal spread and corresponding marginal diminution of the density of the current.

The fact that the current applied in electroshock travels along a fairly straight path from one contact to the other is also overwhelmingly proved by clinical experience. The danger of occipitally placed electrodes, amply confirmed by Alpers and Hughes⁶ in animal experiments, is explained by the fact that in that placement, a significant amount of current passed through the vasomotor centers of the hindbrain, which does not occur with bifrontally placed electrodes, or to a clinically troublesome degree with temporo-parietal placement of the leads. Fleischhacker⁹ has also published evidence of the striking and consistent differences of the motor responses in electroshock brought about by only slight asymmetry in the placement of the electrodes on the surface of the skull.

Action currents, of course, irradiate in many directions along neuronal paths excited by the cerebral electrostimulation; these, however, are of a fundamentally different order of magnitude than the shock currents.

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IX

NEUROPHYSIOLOGIC ASPECTS OF PHYSICAL TREATMENTS FOR MENTAL DISEASE

ELECTRIC TREATMENT

Nonconvulsive Treatment (Vasovagal Akinetic Stimulation). Nonconvulsive, or better vasovagal akinetic, treatment is administered by unidirectional currents ranging from $\frac{1}{2}$ to 10 milliamperes of average current, with spikes ranging from 70 to 252 milliamperes. It can be administered with a variety of machines, provided that they are suitable for increasing and lowering the current by a dial with the control of an amperemeter, in order to find and stay within the desired amount of current adapted to the patient's stimulation thresholds, as observed by his physiologic responses. The physiologic effect of the treatment, of course, depends not only on the basic average current, but also on the magnitude, frequency, and spacing of the spikes as well as on the presence and magnitude of the wave envelope upon which the spikes may be superimposed (see Chapter VIII). The top limit of the spikes varies somewhat among the different models, even between Reiter's two different models, CW 46 and CW 47. In the nonconvulsive range on model CW 46 they rise generally up to 140 times the average current, on model CW 47 up to 39 times (in inverse order to average current level). Furthermore, on model CW 47 the spikes are not superimposed upon a wave envelope, as they are on model CW 46. It is for this reason that with model CW 46, for instance, the effective range of average current lies between $\frac{1}{2}$ and $2\frac{1}{2}$ milliamperes; with the differently patterned less "irritant" current of model CW 47, it is between 2 and 10 milliamperes.

With the Liberson-Offner machine, the effective range for nonconvulsive treatment utilizes an average current of about 2 milliamperes, with peaks up to 80 milliamperes.

In my work with nonconvulsive treatment, I have used both Reiter machines. I prefer the less irritating current of model CW 47 for all kinds of nonconvulsive treatments except that immediately following bifrontal convulsive treatment.

For nonconvulsive treatments, not preceded by convulsive electroshock, and for nonconvulsive treatments immediately following a convulsive treatment through temporoparietal leads. I have used Reiter's model CW 47 exclusively. Such nonconvulsive treatments, as well as those given without preceding convulsive electroshock, are administered through bitemporoparietal leads, through electrodes placed about one inch above the ear

on either side. Since nonconvulsive stimulation at the current levels designated above does not abolish consciousness, such treatments should be initiated under anesthesia—preferably pentothal sodium—administered by the intravenous route; if the treatment is given immediately after a convulsive treatment, the state of postconvulsive anesthesia in the wake of the convulsive treatment suffices for adequate anesthesia, and injection of pentothal sodium can be dispensed with.

The immediate effect of nonconvulsive stimulation through frontal or temporoparietal leads, with a basic unidirectional current of about $\frac{1}{2}$ to

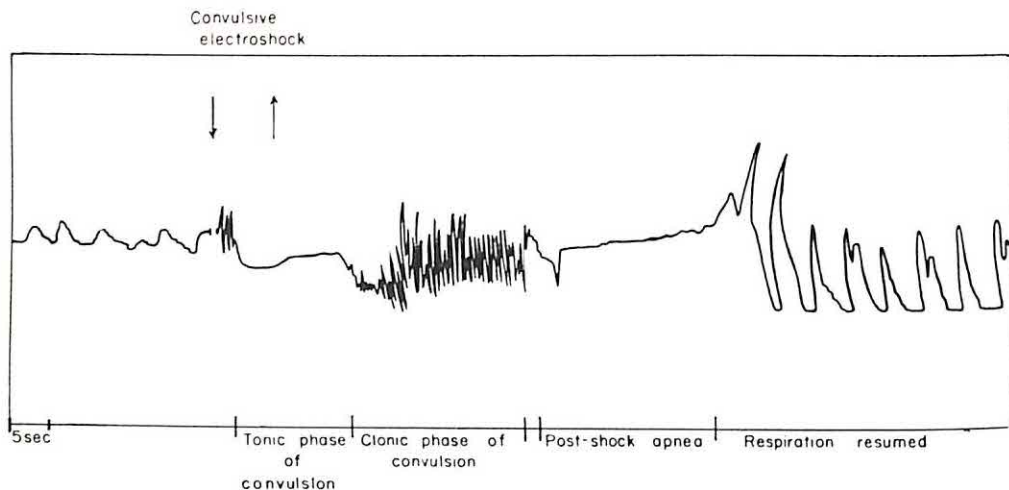


Fig. 12. Kymograph tracing obtained from the patient's chest wall prior to, during, and after a convulsive electroshock treatment. Female patient, age 69 years, suffering from involutional depression.

In this and the following graphs the slow excursions represent respiratory movements; the rapid ones clonic movements, while during tonic contraction and apnea, oscillations are absent, resulting in a smooth, slightly curved, or rather straight line. The arrows in the upper half of the figure indicate the application and duration of the convulsion-inducing electric stimulus, the arrows pointing toward the graph indicating the current being turned on, the arrows pointing away from the graph indicating the current being turned off. The onset and duration of the various phases of the convulsion and the post-convulsive state are indicated at the lower margin of the figure.

Note period of apnea lasting 21 seconds following the clonic phase of the convulsion. (From Alexander, *Am. J. Psychiat.*, vol. 107, 1950.)

$2\frac{1}{2}$ milliamperes, with spikes up to 70 to 98 milliamperes, is stimulation of respiratory activity, which is particularly striking during the apneic phase following a convulsive electroshock or when the patient holds his breath after reaching a state of deep anesthesia under pentothal sodium (Alexander¹). Robie²² and Furst and Reiter⁸ introduced the respiratory stimulant action of nonconvulsive currents as a useful adjunct in the treatment of coma due to barbiturate poisoning. Hoffman and Wunsch¹¹ for resuscitation from insulin coma. Additional immediate effects of nonconvulsive stimulation are slight slowing of the pulse followed by acceleration, slight dilatation of the pupils with bitemporoparietal treatment, slight constriction with bifrontal treatment, the pupils, however, remaining reactive to light throughout the period of stimulation; and, gradually increasing as the stimulation continues, flushing and sweating of the skin, particularly over the face, upper chest, and arms, similar to the distribu-

tion of flush and sweating brought about by an injection of mechohyl, indicating a marked cholinergic (parasympathomimetic) effect of the stimulation.

If the level of current is raised to about 5 milliamperes (with spikes up to 160 MA) on the CW 47 machine, respiration may be suppressed, although sometimes it may continue to be stimulated at this level, the threshold for suppression of respiration being higher. Irrespective of whether or not such currents suppress respiration, there will also appear

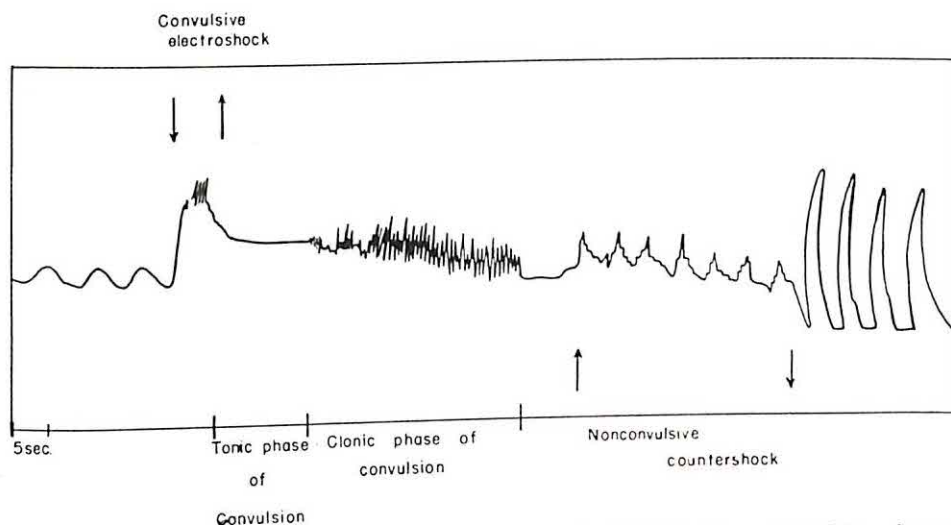


Fig. 13. Kymograph tracing obtained from the chest wall during a convulsive electroshock treatment and an immediately following nonconvulsive countershock treatment of the same patient from whom the recording shown in Fig. 12 was obtained. The arrows in the lower half of the figure indicate the application and duration of the nonconvulsive countershock stimulus, the arrow pointing toward the graph indicating turning on of the nonconvulsive current, the arrow pointing away from the graph indicating turning off of the nonconvulsive current.

Note the immediate resumption of respiratory activity upon initiation of nonconvulsive countershock and its maintenance at a regular rate and amplitude throughout the period of nonconvulsive countershock stimulation. At the same time, the patient's maximally dilated pupils constricted immediately, and her cyanosis was promptly replaced by a bright flush. The electrical stimulus to respiration appears to have precedence over the chemical stimuli; the hyperpnea caused by the accumulation of CO_2 during the treatment does not manifest itself until after the termination of the nonconvulsive countershock stimulation. (From Alexander, *Am. J. Psychiat.*, vol. 107, 1950.)

at this level myoclonic twitchings of the facial musculature, usually only on the side opposite to the positive pole, quickly reversible by reversal of the polarity of the current. These myoclonic twitchings are obviously due to direct stimulation of the cortical facial representation in the motor area, which is the part of the motor area nearest to the electrodes, i.e., at the point of greatest concentration of the current. When this point is reached, the treatment should no longer be classified as nonconvulsive, but as myoclonic treatment, which will be taken up in the next section.

I should like now to give a few examples of the action of nonconvulsive stimulation upon respiratory activity, and then proceed to interpret the phenomena observed from the neurophysiologic point of view. It has long been known that prolonged periods of apnea may occur after a convulsive

electroshock. Figure 12 shows a respirograph tracing obtained from such a patient, showing an apneic phase of 21 seconds' duration, immediately following the clonic phase of the convulsion. If during this apneic phase nonconvulsive treatment is immediately administered as a "countershock" through bifrontal leads at a level of about 1 ($1\frac{1}{2}$ – $1\frac{1}{2}$) milliamperes, with spikes of about 137 (70–160) milliamperes, illustrated in the same patient, who when not treated by nonconvulsive countershock always suffered from fairly long periods of apnea after each treatment, respiratory activity is resumed immediately upon initiation of the nonconvulsive countershock and maintained at a regular rate and amplitude throughout the period of nonconvulsive countershock stimulation (Fig. 13). At the same time, the patient's maximally dilated pupils contract immediately and his cyanosis

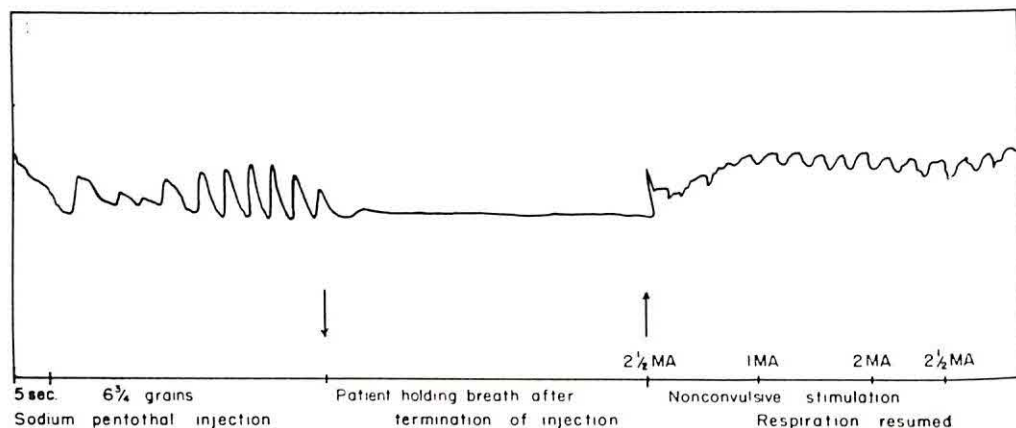


Fig. 14. Kymograph tracing obtained from the chest wall during completion of pentothal sodium anesthesia and beginning of nonconvulsive electric stimulation treatment of a patient aged 28 years, suffering from a schizo-affective psychosis. The arrow pointing away from the graph in the left half of the figure indicates the termination of the pentothal sodium injection; the arrow pointing toward the graph in the right half of the figure indicates the turning on of the nonconvulsive stimulation current.

Note the immediate resumption of respiration upon nonconvulsive electric stimulation. (From Alexander, *Am. J. Psychiat.*, vol. 107, 1950.)

is promptly replaced by a bright flush. The electrical stimulus to respiration appears to have precedence over the chemical stimuli: the hyperpnea caused by the accumulation of CO_2 during the preceding convulsive treatment usually does not manifest itself until after the termination of the nonconvulsive countershock stimulation (Fig. 13).

Figure 14 shows the respirograph record of a patient who was deeply anesthetized by the administration of $6\frac{3}{4}$ grains of pentothal sodium intravenously. As soon as the needle was withdrawn the patient held his breath for 40 seconds. Immediately upon administration of nonconvulsive stimulation at an average level of $2\frac{1}{2}$ milliamperes with spikes up to 98 milliamperes through temporo-parietal leads by means of Reiter's CW 47 machine, respiratory activity was resumed.

Above and below the optimal level of stimulation this respiratory response to nonconvulsive electric stimulation becomes paradoxical and ultraparadoxical respectively, indicating that this respiratory effect conforms to Pavlov's laws of higher cortical activity (Pavlov,²⁰ Konorski¹²). At its highest immediately subconvulsive levels, but definitely below the

minimum threshold necessary to produce a tonic convulsive reaction in a given individual, nonconvulsive stimulation will inhibit or arrest respiration, in the manner of Pavlov's paradoxical reaction. If subsequently the current is lowered to about half its former strength, respiration will immediately be stimulated, in the manner of Pavlov's ultraparadoxical reaction. Figure 15 is the respirograph record of a nonanesthetized patient in whom stimulation at the average level of $12\frac{1}{2}$ milliamperes with spikes up to 300 milliamperes inhibited respiration, without initiating a tonic or other convulsive reaction apart from slight myoclonic twitching of the facial musculature. Lowering of the current to an average of 5 milliamperes, with

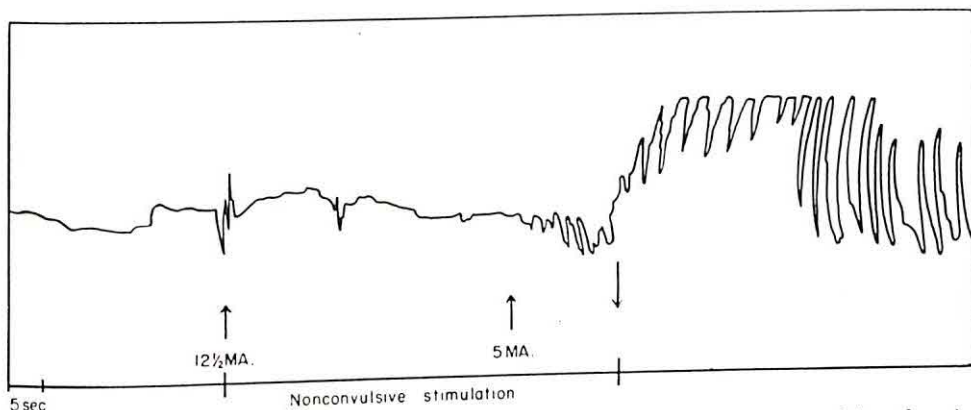


Fig. 15. Kymograph tracing obtained from the chest wall during nonconvulsive electric stimulation treatment without anesthesia in the case of the same patient from whom the recording shown in Fig. 14 was obtained. The first arrow pointing toward the graph indicates the turning on of the initial stimulating current (average current of $12\frac{1}{2}$ milliamperes), the second arrow pointing toward the graph indicates the point at which the stimulating current was lowered to 5 milliamperes. The third arrow, pointing away from the graph, indicates the termination of the electric stimulus.

Note that for the first 40 seconds, nonconvulsive stimulation was at immediately subconvulsive level, sufficient to inhibit respiration; lowering of the current to less than one-half during the last 15 seconds of treatment promptly restored respiration within $2\frac{1}{2}$ seconds after lowering the current. (From Alexander, *Am. J. Psychiat.*, vol. 107, 1950.)

spikes up to 160 milliamperes, promptly stimulated respiration. Sometimes a current of 5 milliamperes (with spikes up to 160 milliamperes) will inhibit respiration; lowering the current to $2\frac{1}{2}$ milliamperes (with spikes up to 98 milliamperes) will promptly stimulate respiration. On a few rare occasions I found even a current of $2\frac{1}{2}$ milliamperes (with spikes up to 98) inhibitory to respiration; respiration was promptly stimulated, however, when the current was lowered to 1 milliampere (with spikes up to 50 milliamperes). These facts are not only of theoretical interest, but of practical importance for the safe administration of nonconvulsive electric stimulation therapy.

Yanoff,²⁷ utilizing my method of nonconvulsive suppression of respiration, made a most important observation. He found that whenever respiration is suppressed at threshold values (for instance, at 5 milliamperes of average current on Reiter's CW 47 machine) and suppression continued at that level, this suppressive effect usually does not last longer than 40 seconds. Irrespective of the continuation of the stimulus, respiration is resumed at about that time. This phenomenon is of particular theoretical in-

terest, because the time of about 40 seconds has been found to be the average duration of a major tonic-clonic seizure as well (Alexander and Löwenbach, ref. 4, p. 93. Sulzbach, Tillotson, Guillemin and Sutherland.²⁴ Alexander²).

This parallelism may indicate that nonconvulsive stimulation produces not only direct and immediate stimulant and inhibitory effects upon the

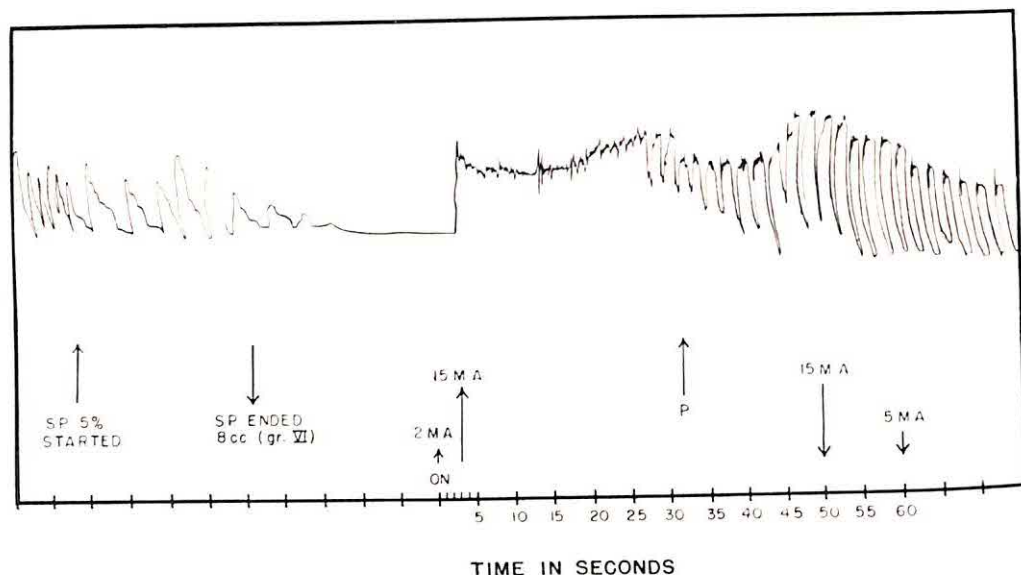


Fig. 16. Kymograph tracing obtained from the patient's chest wall during myoclonic stimulation treatment. The first arrow on the left pointing toward the graph indicates the start of the pentothal sodium injection. The next arrow, pointing away from the graph, indicates the termination of the pentothal sodium injection 23 seconds later. The next small arrow pointing toward the graph indicates turning on of the nonconvulsive stimulation test current (2 milliamperes of average current) 24 seconds later. The next arrow indicates the raising of the stimulating current to the myoclonic range (15 milliamperes of average current) 3 seconds later. The next arrow pointing upward indicates change in polarity (marked by P) 29 seconds later. The next arrow pointing away from the graph (50 seconds after the start of the electric stimulation) indicates the point at which the stimulating current is begun to be gradually reduced to 5 milliamperes within 10 seconds, as indicated by the last arrow pointing away from the graph (60 seconds after the start of the stimulation).

Note that the myoclonic seizure—apnea with superimposed myoclonic twitches—produced by stimulation with an average current of 15 milliamperes ceases after 24 seconds, at which time respiratory movements are resumed. Minute myoclonic spikes, however, still remain superimposed over the respiratory movements until the current is reduced. At the level of 5 milliamperes of average current a single spike superimposed over each inspiratory movement indicates a slight degree of laryngospasm, which disappeared within 15 seconds after the current was reduced further, in this case to 4 milliamperes of average current, 38 seconds later (not shown in this graph).

nerve tissue of the brain, but also sets off an actual seizure, to be sure, not a major tonic-clonic one, but a seizure nevertheless. Such seizures have their parallels in epilepsy; epileptologists classify them as vasovagal seizures (Schwab²³). For this reason, I feel that the most precise term for the type of stimulation described above is the term "vasovagal akinetic stimulation."

Myoclonic Treatment (Sub-Grand Mal Convulsive Stimulation). If the current on Reiter's CW 47 machine or the Liberson-Offner machine is

raised to slightly below the threshold necessary to set off a major grand mal convulsion, myoclonic rhythmic twitchings make their appearance, beginning in the face at levels close to the nonconvulsive level, and then involving, as the current is raised, first the arms and hands, then the trunk, and finally the legs. These myoclonic twitchings start and are first always more marked on the side opposite to the positive pole of the current, and are obviously due to direct stimulation of the motor area of the cortex of the brain. For the universal production of myoclonic twitchings all over the body, an average current of 10 to 15 milliamperes (with spikes up to 250 to 345 milliamperes) is necessary, if the treatment is given with the CW 47 machine and the No. 1 current pattern used.

The march and pattern of these myoclonic twitchings are similar to a Jacksonian attack. Like any other seizure, these electrically induced myoclonic Jacksoniform seizures are self-limited as to time; even if the stimulation is continued (at the same threshold value), the seizure ends after about 20 to 45 seconds and normal respiration is resumed (Fig. 16), provided that the stimulation was applied at the right level, that is, a level of current below that capable of setting off a major grand mal convulsion.

The myoclonic twitchings stop immediately whenever the electric stimulus is discontinued, i.e., the subject is at no time irrevocably committed to the convulsion, and termination of the stimulus at any time leaves the subject limp. This is in diametric contrast to a grand mal convulsion, in which termination of the stimulus during the convulsion itself does not terminate the convulsion, which goes on irrevocably through its tonic and clonic phases. The duration of a major grand mal convulsion is not in any way influenced by the cessation of the convulsion-inducing stimulus, while the myoclonic convulsion ceases as soon as the electric stimulus is discontinued.

Convulsive Treatment (Grand Mal-Producing Stimulation). *Duration and Pattern of the Convulsion.* A major convulsion induced electrically is a motor event which is sharply limited in time. Once the convulsive threshold has been reached, the duration of the convulsion is independent of the strength and duration of the convulsion-inducing stimulus.

The range of duration of convulsions occurring spontaneously as in epilepsy is wider than that of electrically induced convulsions. Oppenheim¹⁹ states that the tonic phase may last from a few seconds to one-half minute, the clonic phase from one-half minute to a maximum of 5 minutes.

Alexander and Löwenbach (p. 93) found that electrically induced convulsions in cats (with shocks of 5 seconds' duration) were far more uniform in timing: we found them in the vast majority of instances to vary from 35 to 39 seconds, irrespective of the intensity of the convulsion inducing current. In these studies, however, we did not concern ourselves with the relative duration of the two main phases. Myerson, Feldman and Green¹⁸ found that electrically induced convulsions, like those induced by metrazol, last 30 to 45 seconds. Sulzbach, Tillotson, Guillemin, and Sutherland²⁴ found that convulsions induced by alternating current of 500 milliamperes of 0.4 second's duration lasted an average of 38 seconds, the range extending from 23 to 55 seconds. They found the latency period prior to the onset of these convulsions to vary from 0.1 to 24.9 seconds, the average being 1.8 seconds. They did not give figures for the relative duration of the tonic and clonic phases.

lus was below the convulsive threshold of the individual, or because that threshold was raised artificially, for instance, by anticonvulsant drugs. Nevertheless, this brief period of tremulous tetanic activity is present irrespective of whether it leaves the subject limp immediately or whether it is followed by a major convulsion. It seemed useful for a study of this type to differentiate sharply between the build-up period during which the subject is not yet committed to the total march of events characteristic of the convulsion on the one hand, and the convulsion itself, during which the subject is committed to a definite march of events, on the other. I therefore called the former phase "latency," the latter, the convulsion itself, subdivided into its tonic and clonic phases. This differentiation aided

Table 2. Duration in Seconds

NO. OF CASES	TYPE AND STRENGTH OF CURRENT	STIMULUS	LATENCY	TONIC PHASE	CLONIC PHASE	ENTIRE CONVULSION	TOTAL WITH LATENCY
20	Alternating current 924 MA peak to peak	0.5	2	9	24	33	35
28	Unidirectional current with spikes up to 925 MA	5	5	9	33	42	47
19	"	45	9	31	11	42	51
33	"	47	10	34	8	42	52
18	"	48	9	39	4	43	52
7	"	54	11	43	0	43	54

the clarification of the phenomena under investigation, especially in view of the fact that the latency phase varied greatly with the amount and duration of the electric stimuli, while the time span of the convulsion itself showed very little variation.

The total time of the motor event resulting from the electric stimulation with alternating currents as specified above, including the convulsion and the preceding latency period, lasted an average of 35 seconds. It is of interest that this time span of the convulsive seizure is practically identical with that which Löwenbach and I had established for the cat, using the same type of alternating current; in these earlier studies we measured the time from the application of the stimulus to the termination of the resulting convulsion. We made no separate allowance for the latency period which elapsed between them.

When unidirectional current of 8 to 15 milliamperes with spikes up to 540 to 925 milliamperes of 5 seconds' duration was used, the period of latency preceding the onset of the tonic phase became significantly prolonged: it was an average of 5 ($2\frac{1}{2}$ to 10) seconds (Fig. 18, Table 2). The tonic phase, however, remained unchanged, namely, 9 seconds (varying from 4-13 seconds). The clonic phase, on the other hand, was longer; it lasted from 21 to 39 seconds, the average being 33 seconds. This longer

clonic phase caused the entire convulsion to be of slightly longer duration, namely from 26 to 50 seconds, the average duration being 42 seconds (Table 2).

Attempts to understand the nature of the tonic and the clonic phases of the convulsion have occupied neurologists' investigative efforts for some time. A generally accepted view of their physiologic significance and interrelationship, however, has never been formulated. The most recent theory is that of Penfield and Erickson,²¹ who regarded the clonic phase as arising in the cortex, the tonic phase as arising in the subcortical gray matter. Walshe²⁶ disputed this theory on the grounds that in convulsions be-

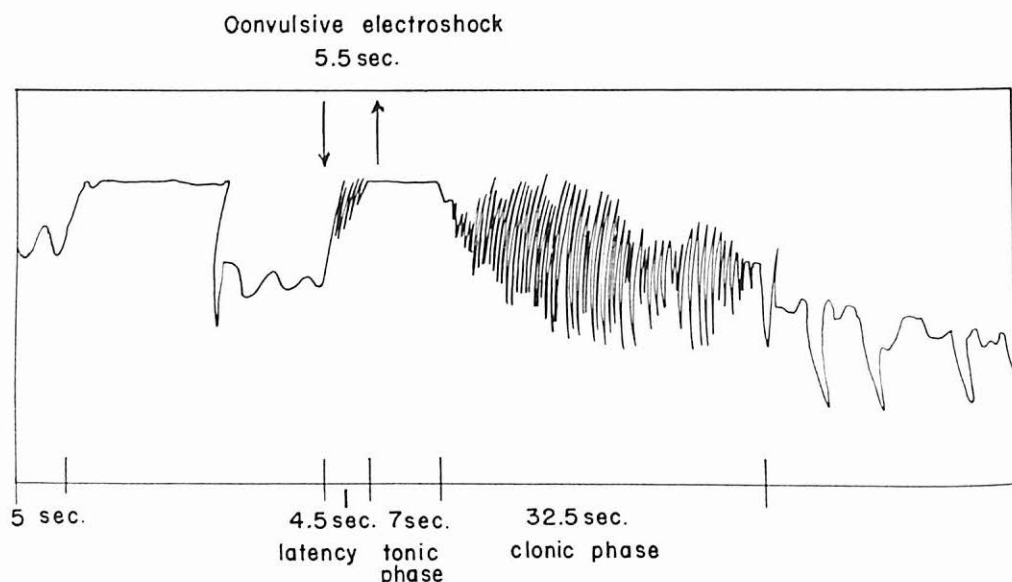


Fig. 18. Convulsive electroshock treatment with unidirectional current (average current of 15 milliamperes with spikes up to 925 milliamperes) of 5.5 seconds' duration. (From Alexander, J. *Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

ginning unilaterally tonic spasm not rarely precedes clonic, and in generalized convulsions such as in epilepsy the latter invariably does so. Walshe states that it may be said in general that a convulsion that remains restricted to a narrow field of musculature and is prolonged tends to be clonic from the outset, while a rapidly spreading convulsion may be tonic for a few moments before the spasm begins to intermit, that is, to become clonic.

Suppression of the Clonic Phase by Continued Stimulation. Tietz and her coworkers²⁵ were the first to discover that the motor phenomena of the convulsion could be significantly modified by modification of the current level and by the duration of current flow. They found that by raising the current to its convulsive level gradually, maintaining it for approximately 30 seconds and then lowering it gradually, the violence of all the motor phenomena, including those of the initial tetanic contraction, the tonic phase, and the clonic phase, could be greatly reduced. In particular the clonic phase could thus be greatly diminished or virtually eliminated. These authors, however, did not determine the duration of the various phases of the convulsion, nor did they attempt to eliminate the clonic phase

entirely. I therefore decided to study this problem with a method allowing automatic recording and precise measurement of time relationships and to determine whether or not the clonic phase could be suppressed entirely by raising the time of application of unidirectional current to the full time span of the convulsion, that is, up to 60 seconds (Alexander²).

This study was carried out by kymograph recordings obtained from the patient's chest wall. Eighty-one convulsive treatments in 27 different patients were thus recorded. One thousand eighty-three additional convulsive treatments of the same type were carried out in 154 additional patients; the quantitative data, however, were entirely based on the first series of 81 consecutive convulsive treatments recorded by kymograph (Table 2).

The alternating current treatments used as controls were administered by means of the standard Medcraft and Rahm machines, the unidirectional current treatments by means of the Reiter CW46 machine. All treatments were given through uniformly placed bifrontal leads. Peak to peak currents delivered by the alternating current machines measured up to 924 milliamperes, average currents up to 330 milliamperes. The alternating current treatments with the Medcraft machine were given with glissando attachment, developed according to the glissando technique of Tietz and her coworkers.²⁵

The treatments on the Reiter CW46 machine were also given with Tietz's glissando technique, that is, currents were raised within 1 to 2 seconds from as low as an average current of 3 to 4 milliamperes (with spikes up to 270 to 320 milliamperes) to maximal levels varying from 5 to 15 milliamperes of average current with spikes of from 370 to 925 milliamperes. The current values given in Table 2 represent, of course, the maximal currents, which were uniformly attained within 1 to 2 seconds.

It was found that prolongation of the convulsion-inducing electric stimulus prolonged the period of latency prior to the onset of the major convulsion as well as the relative duration of the tonic phase without a change in the total time span of the convulsion.

Prolongation of the convulsion-inducing stimulus, using unidirectional current of the same strength, prolonged the period of latency to an average of 10 seconds (2-33 seconds) for stimuli of an average of 47 (23-60) seconds' duration (Table 2). The duration of the entire convulsion itself, however, remained entirely unaffected by the longer duration of the convulsion-inducing stimulus: it remained an average of 42 (27-61) seconds for all prolonged stimuli varying from 23-60 (average 47) seconds, and 43 (31-61) seconds for stimuli averaging 48 and 54 (40-60) seconds respectively (Table 2). The duration of the clonic phase, however, was significantly reduced, because it was usually entirely suppressed for the duration of the convulsion-inducing stimulus and only emerged during the balance of the time span of the convulsion, the convulsion remaining entirely tonic as long as the convulsion-inducing stimulus was continued. If the convulsion-inducing stimulation was continued throughout the time span of the convulsion (about 43 seconds) and the latency period (about 11 seconds) that is, on the average for a total of 54 seconds, the clonic phase could be completely suppressed, and the convulsion remained entirely tonic (Table 2). I should like to give a few examples: a convulsion-producing stimulus applied for 37 seconds produced a tonic convulsion after a latency period of 14 seconds. The convulsion remained tonic for the remaining 23 seconds of the stimulus, then became clonic for the remaining 16 seconds of the time span of the convulsion lasting a total of 39 seconds

(Fig. 19). A convulsion-producing stimulus applied for 45 seconds produced a tonic convulsion after a latency period of 8 seconds. The convulsion remained

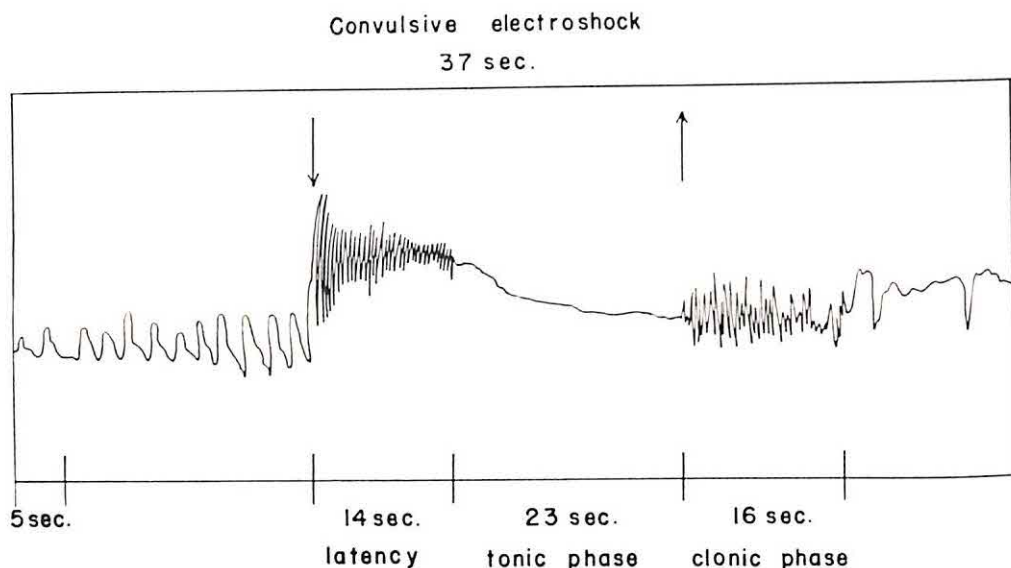


Fig. 19. Convulsive electroshock treatment with unidirectional current (average current of 15 milliamperes with spikes up to 925 milliamperes) of 37 seconds' duration.

Note prolongation of tonic phase, its end coinciding with the termination of the electric stimulus. (From Alexander, *J. Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

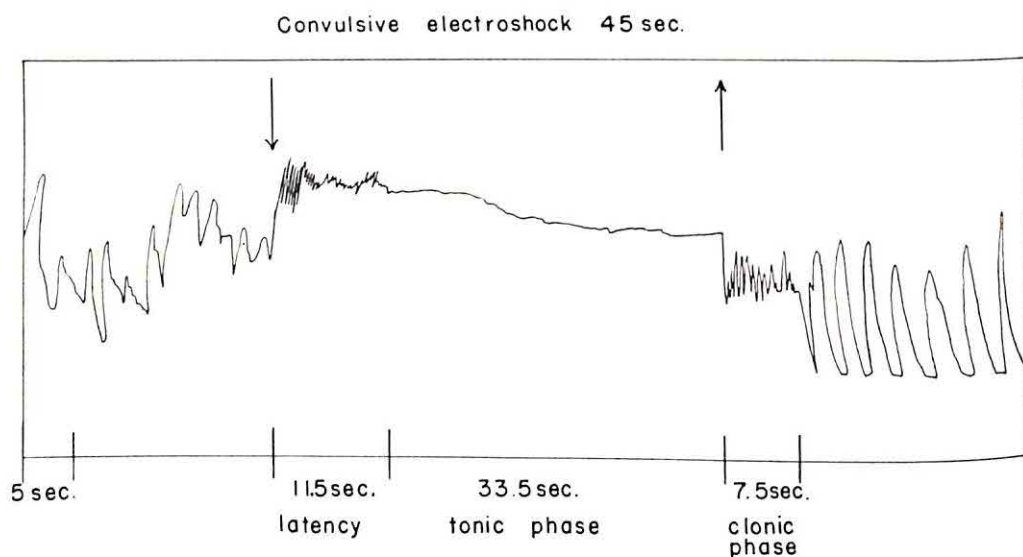


Fig. 20. Convulsive electroshock treatment with unidirectional current (average current of 6 milliamperes with spikes up to 420 milliamperes) of 45 seconds' duration.

Note further prolongation of tonic phase, its end coinciding with the termination of the electric stimulus, leaving only $7\frac{1}{2}$ seconds of the total time span of the convulsion for the clonic phase. (From Alexander, *J. Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

tonic for the remaining 37 seconds of the stimulus, then became clonic for the remaining 6 seconds of the time span of the convulsion lasting a total of 43 seconds. In another case a stimulus of the same duration produced a tonic convulsion after a latent period of $11\frac{1}{2}$ seconds. It remained tonic for the

remaining $33\frac{1}{2}$ seconds of the stimulation, then became clonic for the remaining $7\frac{1}{2}$ seconds of the time span of the convulsion which was 41 seconds in this case. (Fig. 20.)

The clonic phase was reduced to as little as $2\frac{1}{2}$ seconds in another case (Fig. 21). In this case a stimulus of the same duration (45 seconds) produced a tonic convulsion after a latent period of 2 seconds, the convulsion remaining tonic for $43\frac{1}{2}$ seconds, that is, only $\frac{1}{2}$ second longer than the stimulus, while it became clonic for the last $2\frac{1}{2}$ seconds of its total time span, which was 46 seconds in this case.

With stimuli above 52 seconds it was possible to suppress the clonic phase entirely in the vast majority of cases, because such stimuli either

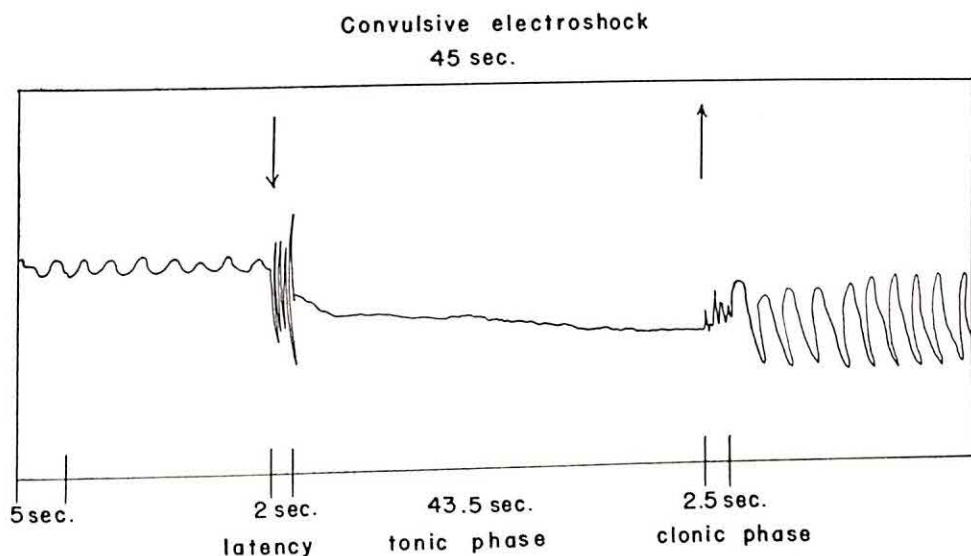


Fig. 21. Convulsive electroshock treatment with unidirectional current (average current of 5 milliamperes with spikes up to 370 milliamperes) of 45 seconds' duration.

Note that the convulsion remains almost entirely tonic. Only the final $2\frac{1}{2}$ seconds of the time span of the convulsion left after the termination of the electric stimulus remain for the manifestation of the clonic phase. (From Alexander, J. *Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

coincided with or outlasted the natural average time span of the convulsion including the latency period.

A stimulus applied for 54 seconds, for instance, produced a tonic convulsion after a latency period of 6 seconds. It remained tonic for the entire period of the convulsion which lasted 48 seconds in this case, its end coinciding with the termination of the stimulus. Immediately upon termination of this purely tonic convulsion the body of the patient relaxed and respiration was resumed without an intervening period of apnea during the "limp" postconvulsive phase. The same result was obtained after a stimulus of 60 seconds' duration (Fig. 22). Although the latency period was unusually long in this case, lasting 17 seconds, the time span of the convulsion, which was 43 seconds in this case, in addition to the latency period of 17 seconds, neatly coincided with the duration of the electric stimulus, thus allowing no additional time for a clonic phase.

In some cases the total time span of the convulsion including the latency period was short enough to allow complete suppression of the clonic phase by

stimuli as short as 40 or 45 seconds respectively. Such a case is illustrated in Fig. 23. In this case a convulsion-producing stimulus applied for 45 seconds

Convulsive electroshock

60 sec.

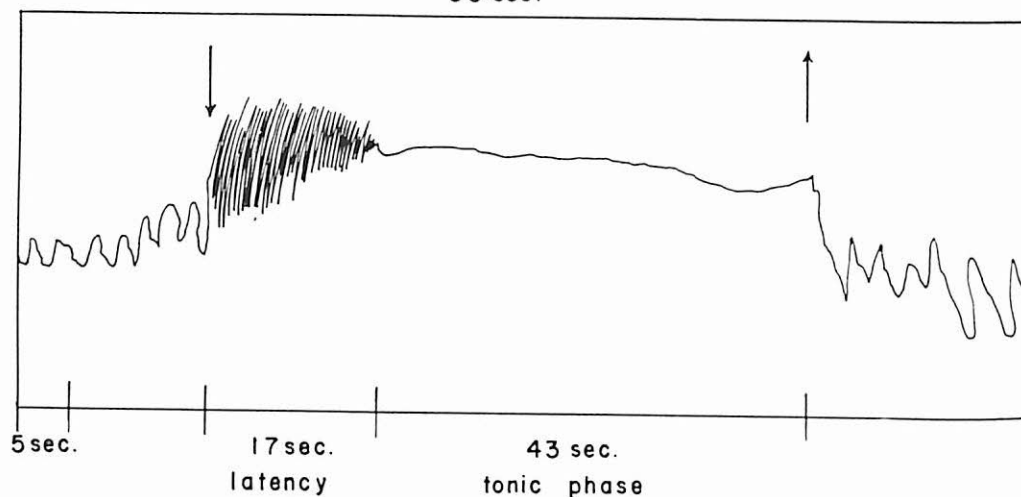


Fig. 22. Convulsive electroshock treatment with unidirectional current (average current of 15 milliamperes with spikes up to 925 milliamperes) of 60 seconds' duration.

Note complete abolition of clonic phase, the end of the time span of the convulsion throughout its time span of 43 seconds, its end coinciding with the termination of the electric stimulus. (From Alexander, J. *Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

Convulsive electroshock

45 sec.

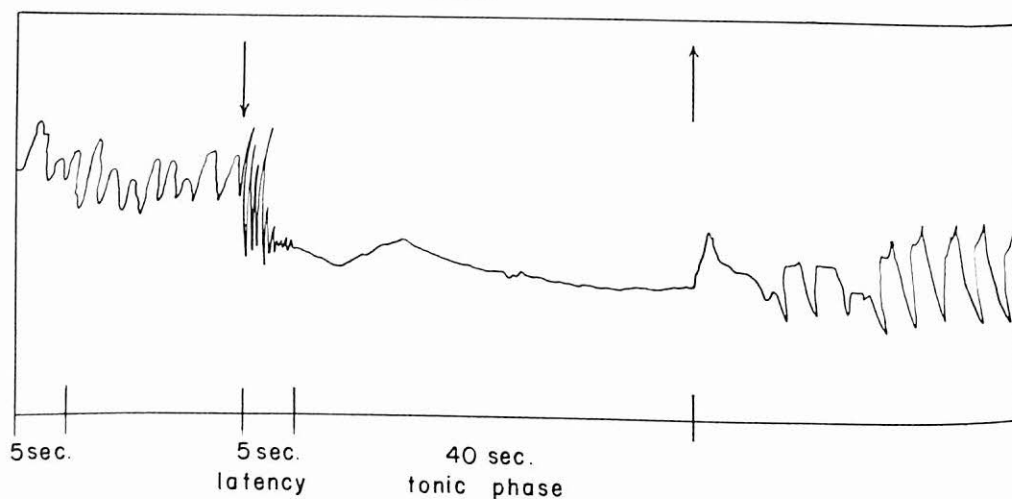


Fig. 23. Convulsive electroshock treatment with unidirectional current (average current of 6 milliamperes with spikes up to 420 milliamperes) of 45 seconds' duration.

Note complete abolition of clonic phase, the end of the time span of the convulsion coinciding with the termination of the electric stimulus. (From Alexander, J. *Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

produced a tonic convulsion after a latency period of five seconds. The convulsion remained tonic for its entire time span, which happened to be forty seconds in this case. Its natural termination thus coincided with the termina-

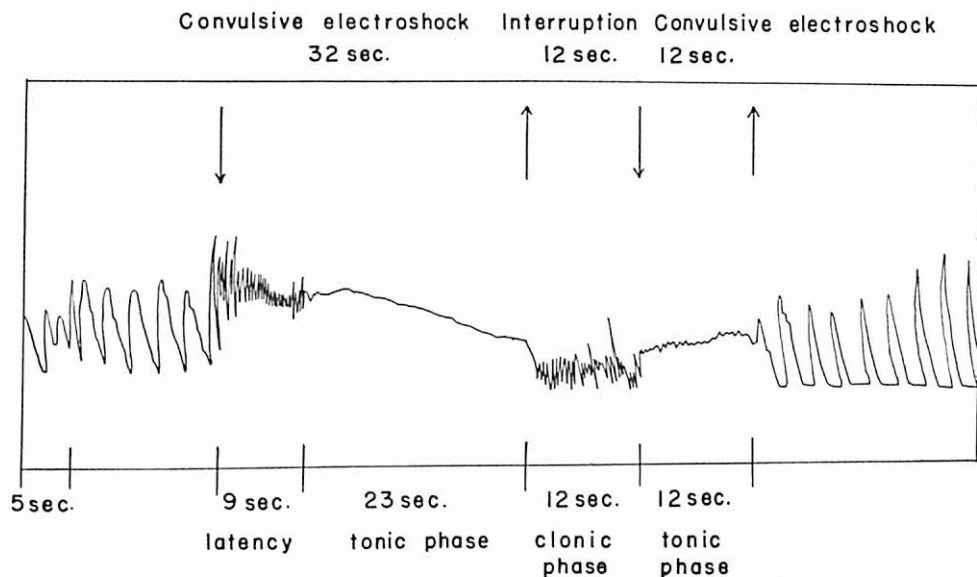


Fig. 24. Convulsive electroshock treatment with unidirectional current (average current of 7 milliamperes with spikes up to 490 milliamperes), with interruption of current for 12 seconds after the first 23 seconds of the convulsion.

Note suppression of clonus during the first 23 seconds of the convulsion and during the last 12 seconds of the convulsion, that is, whenever electric current was flowing, but immediate eruption of clonus during the intervening 12 seconds when electric stimulation was interrupted. (From Alexander, J. Neuropath. & Exper. Neurol., vol. 11, 1952.)

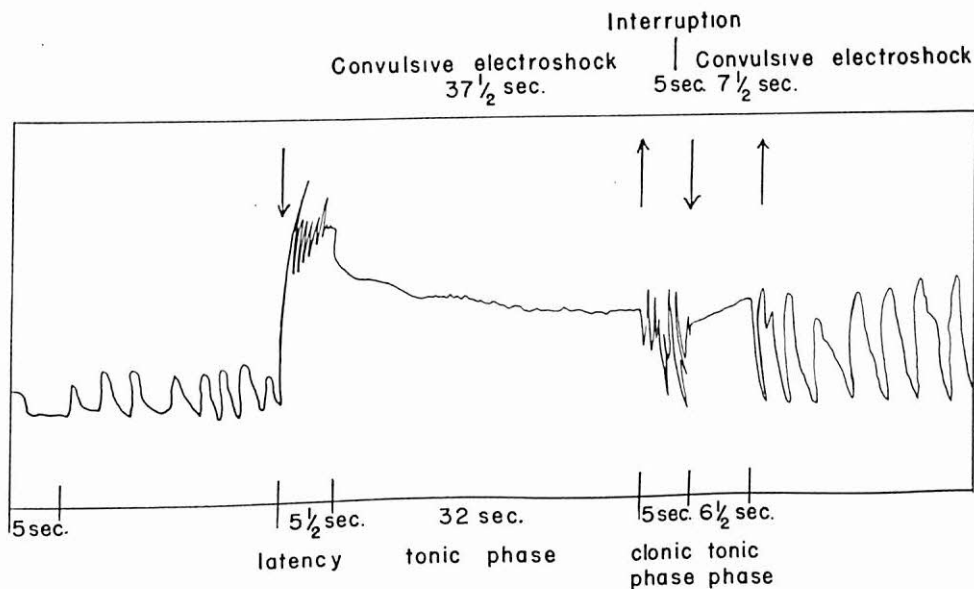


Fig. 25. Convulsive electroshock treatment with unidirectional current (average current of 6 milliamperes with spikes up to 420 milliamperes), with interruption of current flow for 5 seconds after the first 32 seconds of the convulsion.

Note abolition of clonus during the first 32 seconds of the convulsion and during the last 6½ seconds of the convulsion while electric current was flowing, but immediate eruption of clonus during the intervening 5 seconds when electric stimulation was interrupted. (From Alexander, J. Neuropath. & Exper. Neurol., vol. 11, 1952.)

tion of the convulsion-producing stimulus. At the end of this purely tonic convulsion, as soon as the current was turned off, the patient's tone relaxed and he immediately resumed respiration, without an intervening clonic phase.

Whenever the electric stimulation was interrupted during the course of what normally would have been the clonic phase, namely during the latter two-thirds of its time span, violent clonic movements immediately manifested themselves and persisted as long as the stimulus was off; for instance, 12 seconds (Fig. 24), five seconds (Fig. 25), or three seconds (Fig. 26) respectively. They could, however, immediately be suppressed again by reapplying the electric stimulus (Figs. 24, 25, and 26).

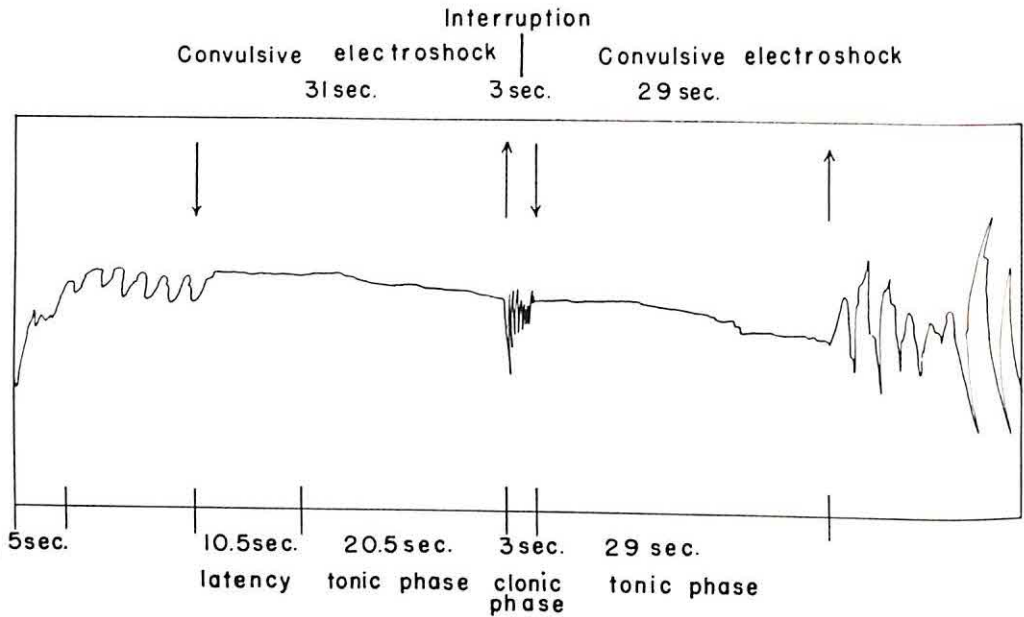


Fig. 26. Convulsive electroshock treatment with unidirectional current (average current of 7 milliamperes with spikes up to 490 milliamperes), with interruption of flow of current for 3 seconds after the first 20.5 seconds of the convulsion.

Note complete abolition of clonus throughout most of the convulsion except for the period of 3 seconds halfway through the convulsive treatment when the current was interrupted for 3 seconds. (From Alexander, *J. Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

In a few cases slight clonic movements came through in spite of non-interruption of the stimulus. These could readily be suppressed by slightly raising the stimulating current (Fig. 27).

In nineteen cases in which stimulation was continued for 45 seconds, the clonic phase could be reduced to an average of 11 (2-32) seconds. If all cases with long continued stimulation are included, averaging 47 (23-60) seconds, the clonic phase was reduced to an average of 8 (0-32) seconds. If only those 18 cases are included in which no clonus broke through during the period of stimulation lasting an average of 48 (40-60) seconds, the clonic phase appeared reduced to an average of 4 (0-21) seconds. For complete suppression, that is, reduction to zero, of the clonic phase, an average time of stimulation lasting 54 seconds (40-60 seconds) was necessary (Table 2). The intensity of current necessary to suppress the clonus during the seizure is the same or slightly less than that necessary to produce the seizure. Lowering of the currents used in this study by as little as one-eighth of the amperage will sometimes allow the clonus to break through to a slight degree, while at three-fourths to

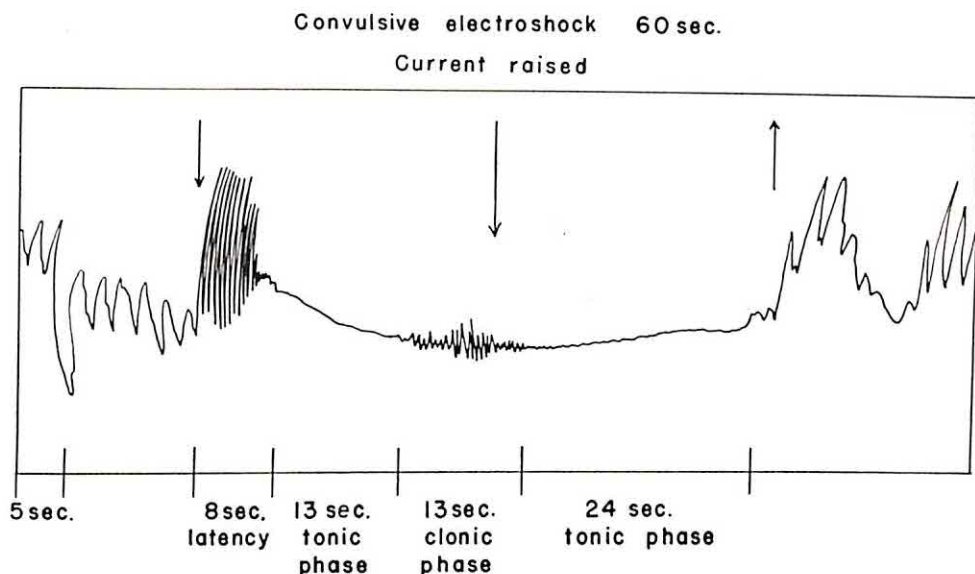


Fig. 27. Convulsive electroshock treatment with unidirectional current of 60 seconds' duration, induced with average current of 6 milliamperes with spikes up to 420 milliamperes flowing for 31 seconds, then raised to an average current of 15 milliamperes with spikes up to 925 milliamperes flowing for the remaining 29 seconds.

Note emergence of clonus about halfway through the treatment, after 21 seconds of flow of current, promptly abolished by raising the current to an average current of 15 milliamperes with spikes up to 925 milliamperes, 10 seconds later. (From Alexander, *J. Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

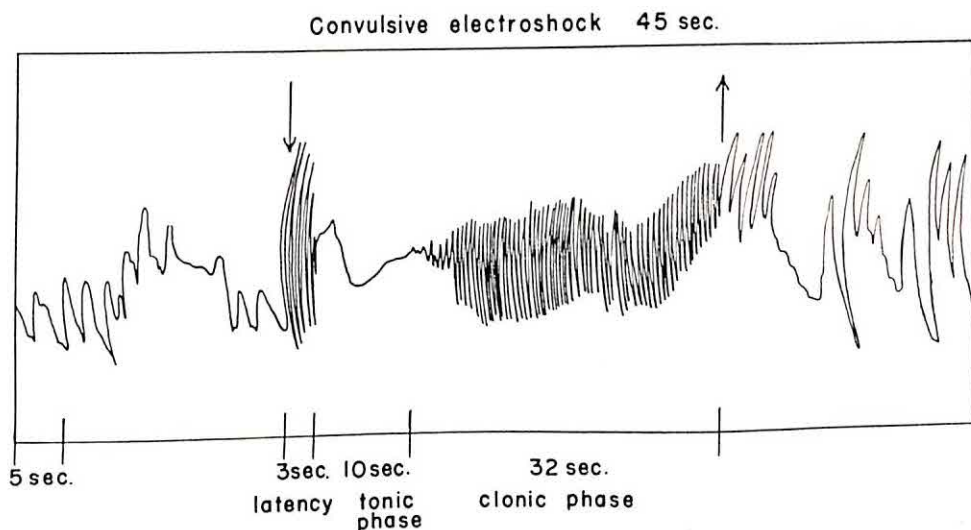


Fig. 28. Convulsive electroshock treatment with unidirectional current (average current of 6 milliamperes with spikes up to 420 milliamperes) of 45 seconds' duration in a woman suffering from hyperthyroidism in addition to depression.

Note emergence of unmodified clonic phase in spite of maintenance of the current throughout the time span of the convulsion. (From Alexander, *J. Neuropath. & Exper. Neurol.*, vol. 11, 1952.)

one-half of the current, clonus will break through regularly, although not with full force. In a few rare instances the current necessary to suppress the clonus is much greater than that necessary to induce the convulsion. In such cases, unless the current can be raised promptly upon the appearance of the first few clonic bursts, the clonic phase will manifest itself in spite of the maintenance of the convulsion-inducing stimulus throughout the time span of the convulsion (Fig. 28). One of the few cases in which I had occasion to observe this phenomenon was a case of a depression in a patient suffering also from hyperthyroidism (Fig. 28).

Excitation and Inhibition in Electroshock Treatment; Respiration as an Indicator of Cortical Activity. In interpreting the phenomenon of the suppression of the clonic phase two alternate possibilities had to be considered: whether this purely tonic convulsion produced under the influence of continued cortical stimulation represented an extreme or "tetanic" excitation of the cortex, or whether it was due to inhibition of cortical activity during the convulsion under the influence of a stimulus presumed to have become inhibitory after its initial excitatory effect. Clinical observation presented indirect evidence in favor of the latter explanation. These clinical observations include the kymographic measurements of the latency period preceding the convulsion and of the apneic phase following the convulsion, observations concerning the disturbance of awareness for memory content after such treatments, and observations concerning the therapeutic effect. The therapeutic effect of purely tonic convulsions upon the psychotic illnesses treated was similar to that of standard types of electroshock treatment, a statistically similar number of treatments being required in both series of cases (6.8 treatments vs. 7.3 treatments per patient). Contrary to expectation, however, the postconvulsive disturbance of awareness produced by purely tonic treatments under prolonged electric stimulation not only did not exceed that produced by standard treatment with its much briefer periods of stimulation by the same type and strength of current, but appeared actually less pronounced.

The most significant finding, however, concerned the occurrence of apnea following the electrically induced convulsion. Following standard treatment in which the clonic phase was allowed to run its course without interference, apnea varying from 6 to 45 seconds occurred in 33 per cent of the cases. In those cases in which the clonic phase was shortened, but not completely eliminated by continued stimulation, the incidence of periods of apnea was reduced to 15 per cent; the apneic phases varied from 6 to 32 seconds. In those cases in which the clonic phase was suppressed entirely, by continuing electric stimulation of the same intensity throughout the time span of the convulsion, no single instance of postconvulsive apnea could be observed at all, respiration starting immediately upon the completion of the convulsion in all of them. Since the postconvulsive apnea is an expression of a fatigue state or refractory phase of the cortex proportional to the intensity of its preceding excitation, the fact that it occurs in inverse ratio to the duration of continued electric stimulation during the convulsion indicates that this continued stimulation does not add to, but rather detracts from the intensity of the cortical excitation during the convulsive seizure, and exerts an inhibiting effect upon the cortical excitation once the seizure has been initiated. This inhibiting effect already expresses itself prior to the start of the convulsion in the

form of prolongation of the average period of latency preceding the beginning of the convulsion.

Higher intensities of current and posterior placement of the electrodes, of course, increase the incidence of apnea in all treated groups; therefore, only currents of similar intensity applied through bifrontally placed electrodes were included in this quantitative comparison.

The following sequence of events emerges from this study: Electrically induced convulsions are initiated by excitation of the cortex, which manifests itself in a short series of bursts of rapid, tremulous tetanic contractions; this fires off the subcortical gray matter, which gives rise to the tonic phase; the subcortical gray matter in turn reexcites the cortex, manifested by the subsequent superimposition of the clonic phase.

Continued electric stimulation probably inhibits the cortex, and thus prevents the reexcitation of the cortex from the subcortical gray matter and abolishes the clonic phase. Since the postconvulsive refractory state of the cortex manifested by apnea and by disturbance of awareness for memory contents is proportional in degree and duration to the preceding state of excitation of the cortex, the prevention of the reexcitation of the cortex, as manifested by abolition of the clonic phase, lessens and shortens its postconvulsive refractory state, thus reducing the incidence of postconvulsive apnea and the degree and duration of postconvulsive disturbance of awareness.

An alternative explanation may be that the effect of such prolonged stimuli as I have used is more purely excitatory in all respects, as exemplified by the purely tonic nature of the contraction without intermittent relaxation, by the prevention of postshock apnea, and by the lessening of subsequent memory disturbance. There is a good deal to be said for either explanation; further studies are needed for the unequivocal theoretical explanation of these phenomena.

Clinical Advantages of the Technique of Suppression of the Clonic Phase. From the general theoretical point of view of understanding the action of electroshock treatment, the finding that abolition of the clonic phase in no way lessened the antidepressant effect of electroshock treatment clearly indicates that the clonic phase is not necessary for the specific antidepressant therapeutic effect of electroshock. The practical clinical value of the technique of suppression of the clonic phase is quite obvious. The lessening of subsequent memory disturbance is the least of these advantages, because that can be brought about by a variety of techniques, including the use of minimal threshold currents (Friedman and Wilcox,⁷ Delmas-Marsalet,⁵ Liberson^{14, 15}) and nonconvulsive countershock, that is, nonconvulsive electric stimulation following the convulsive seizure either immediately or after a time interval (Alexander¹). It is, however, quite apparent to anyone who witnesses the two kinds of treatment—the old standard electroshock and the purely tonic treatment method—that the controlled tonic convulsion is a much gentler procedure. The patient lies quietly in tonic extension, the face is flushed, and respiration begins instantly at the termination of the seizure, or if higher currents are used, can be brought back quickly by shifting to low amperage nonconvulsive stimulation without turning off the current (Alexander¹).

As to the more specific aspects of greater safety, there is no doubt that

the method is capable of greatly reducing, and in skilled hands practically completely preventing, the incidence of postshock apnea and other post-ictal respiratory embarrassment. Furthermore, after 460 consecutive treatments in 80 consecutive patients who have been treated with electrically induced convulsions rendered purely tonic by continued stimulation at the current levels specified in this study,² there were no complaints referable to the spine, nor was there clinical evidence of spinal or other fractures, although cases of osteoporosis in old people, a patient with old spinal injury, and two patients who have suffered spinal fractures during previous courses of standard electroshock treatments have been included. I have not routinely taken x-rays at the completion of each electroshock series, and, therefore, cannot, of course, exclude the fact that asymptomatic fractures may have occurred. In the two cases with old spinal fractures, however, x-rays were taken both before and after the series of controlled tonic electroshock treatments. The x-rays taken at the termination of these treatment series showed neither aggravation of the old fracture nor a new one. A case of a woman six months pregnant has likewise been treated with this method without ill effect to the fetus.

In a subsequent additional series of 704 treatments in 101 patients, the procedure was modified by using higher currents, raising the current from an initial point of 5 milliamperes of average current (with spikes up to 370) to an average treatment current of 20 milliamperes (with spikes up to 1670) in an effort to reduce the period of latency, which, if too long, may sometimes be remembered by the patient. For a long time, no fractures occurred with this modified method either. When I then tried to reduce the period of latency still further by raising the average current from the initial level of 5 milliamperes to the treatment level of 20 milliamperes faster than within 1 second, that is within about one-half second, I produced painful, though otherwise fortunately harmless, spinal fractures (two of them multiple) in 3 patients in fairly rapid succession. It was obvious that these fractures had occurred during the initial tetanic contraction, and the lesson learned from these 3 cases was that this initial contraction should always be softened according to the glissando technique first pioneered by Tietz and her coworkers.²⁵ A full 2 seconds should always be allowed for raising the current, for instance, from an initial average of 5 to a treatment average of 20 milliamperes. Even in fractures caused by the initial tetanic contraction, if and when they occur, especially if they remain minute and subclinical, suppression of the clonic phase probably reduces the tendency to aggravate them by dislocation attributable to violent clonus.

In my practice the above described method of electrical suppression of the clonic phase with currents at minimal convulsive threshold levels (average currents up to 5 milliamperes, with spikes up to 370) has almost completely replaced the use of curare.

This method of continued stimulation not only suppresses the muscular clonus during the clonic phase, but also prevents the generalized vasoconstriction and pilomotor reaction ("goose flesh," *cutis anserina*) which usually manifest themselves during the clonic phase and frequently outlast such uncontrolled convulsions for some time. The reason for this phenomenon is probably that like the clonus itself, this associated vasoconstriction and pilomotor reaction is due to excitation of the cortex, and

therefore suppressed by the inhibitory effect of continued stimulation. Whatever the explanation, the fact remains that whenever the convulsion is rendered purely tonic by continued stimulation, generalized vasodilatation and sweating manifest themselves within the first 30 to 35 seconds of the seizure, increase as the seizure progresses, and outlast the seizure;

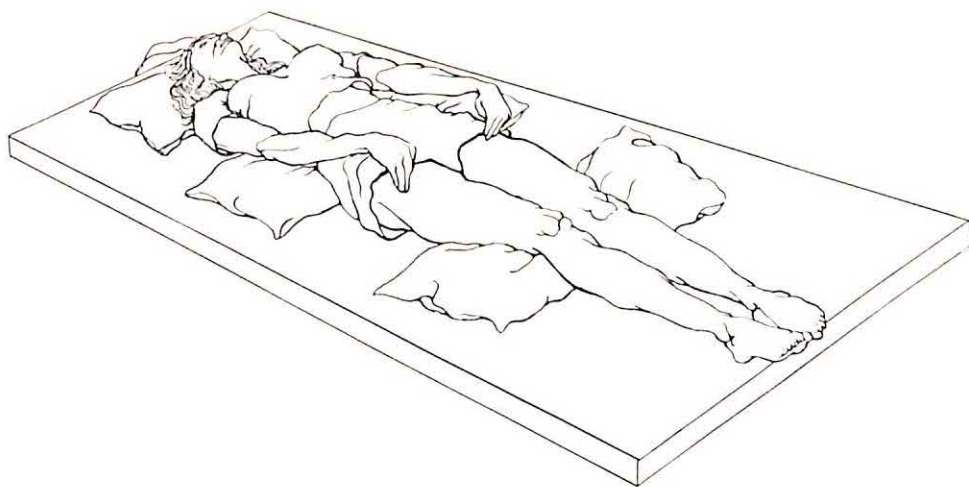


Fig. 29. Extensor type of electrically induced convulsion; note only slight flexion of wrists.

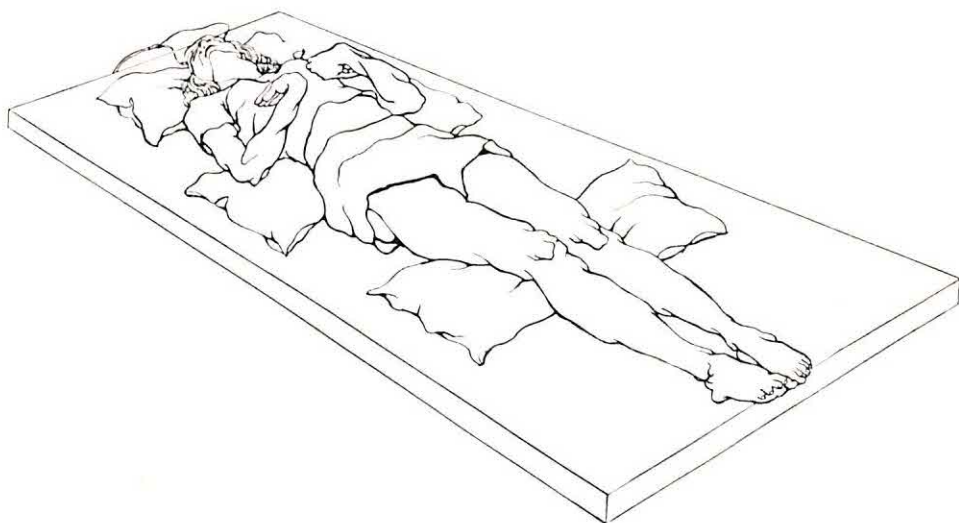


Fig. 30. Arms-flexor, legs-extensor type of electrically induced convulsion.

formerly cold and clammy extremities turn warm, and vasoconstriction and goose flesh remain entirely absent. These observations suggest that the use of such controlled tonic convulsions may be preferable in physical conditions in which generalized vasoconstriction may represent an additional hazard, such as in patients with a history of coronary heart disease or angina pectoris. I have so far treated several such patients with this method successfully and without even temporary ill effect.

As a practical measure, the use of this technique of suppressing the clonic phase has proved useful in preventing postictal respiratory embar-

BIOCHEMICAL CHANGES DURING AND AFTER ELECTRICALLY INDUCED CONVULSIONS



Fig. 32. The relative increase or decrease of the substances and/or ratios enumerated is expressed graphically in the waxing and waning of the black vs. the white part of the disks shown for each period indicated. An equal black and white division of the disk represents the amount or ratio in the state of homeostasis. Increase in the black part indicates relative increase in the substance or the ratio; increase in the white part indicates relative decrease in the substance or the ratio. (From the data of Neustadt et al.³⁹)

rassment as well as the generalized vasoconstriction during and after the seizure, and has in my clinical work almost completely replaced the use of curare in administering electroshock to individuals with abnormalities

of the skeletal system, especially spinal fractures, to pregnant women and in other conditions in which violent clonic convulsions could be harmful.

In treatment-resistant involutional psychoses and in some schizophrenic psychoses, arms-extensor convulsions appear to be more effective than arms-flexor convulsions.

Biochemical Changes Induced by Electroshock Treatment. The possible biochemical links between application of electric current and clinical recovery have been little explored. In a study which I carried out in collaboration with Neustadt, Myerson, Howard, and Kaldeck,³⁹ several groups of biochemical substances were investigated during and after electric shock therapy (Fig. 32). These substances were determined in blood samples drawn simultaneously from the brachial artery, the antecubital vein, and the internal jugular vein by the technique developed by Myerson.^{37,38}

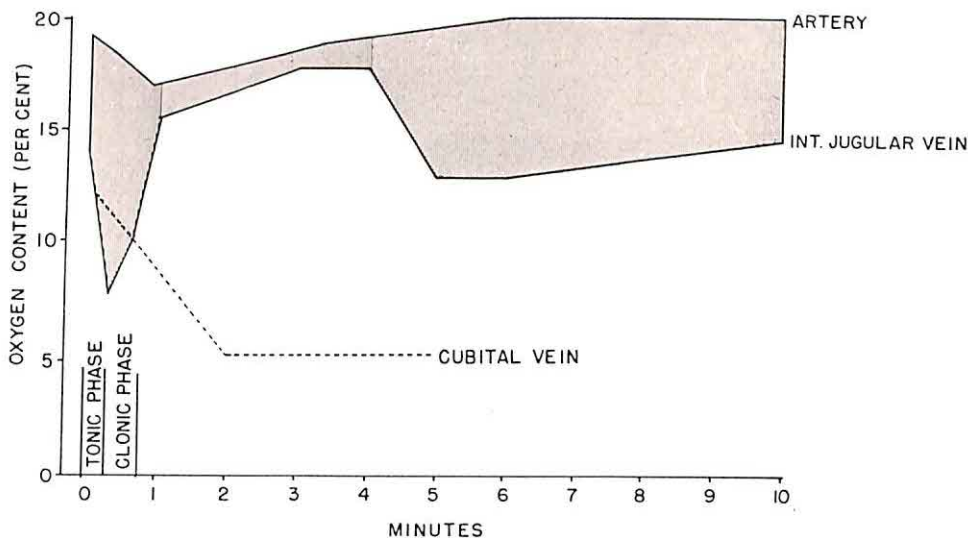


Fig. 33. Oxygen content of arterial and internal jugular venous blood. (From the data of Neustadt et al.³⁹)

The oxygen content of the arterial blood dropped slightly during the tonic phase, and more decidedly during the clonic phase. The oxygen content of the internal jugular venous blood dropped precipitously during the tonic phase, and rose strikingly during the clonic phase and the immediate postconvulsive period (Figs. 32 and 33). Thus during the tonic phase there was a great increase in arteriovenous difference (Figs. 32 and 33), followed by a marked diminution in this value during the clonic phase and the subsequent 3 or 4 minutes of the immediate postconvulsive period. All oxygen values returned to their basic levels within 5 minutes after the start of the convulsion (Figs. 32 and 33).

The carbon dioxide content dropped and remained low for about 30 minutes after treatment (Fig. 32).

The blood sugar rose continuously for at least one-half hour (Figs. 32 and 34). Cholinesterase dropped about 50 per cent during the first 3 minutes, and remained low for 45 to 90 minutes (Figs. 32 and 35). Royer and his collaborators⁴¹ confirmed this observation. Creatinine did not change in the arterial or peripheral venous blood during and after electro-

shock in 80 per cent of all subjects. However, in 90 per cent of all subjects, a surprising increase of creatinine was found in the internal jugular venous blood (Fig. 32). This rise was 20 to 30 per cent of the initial value and

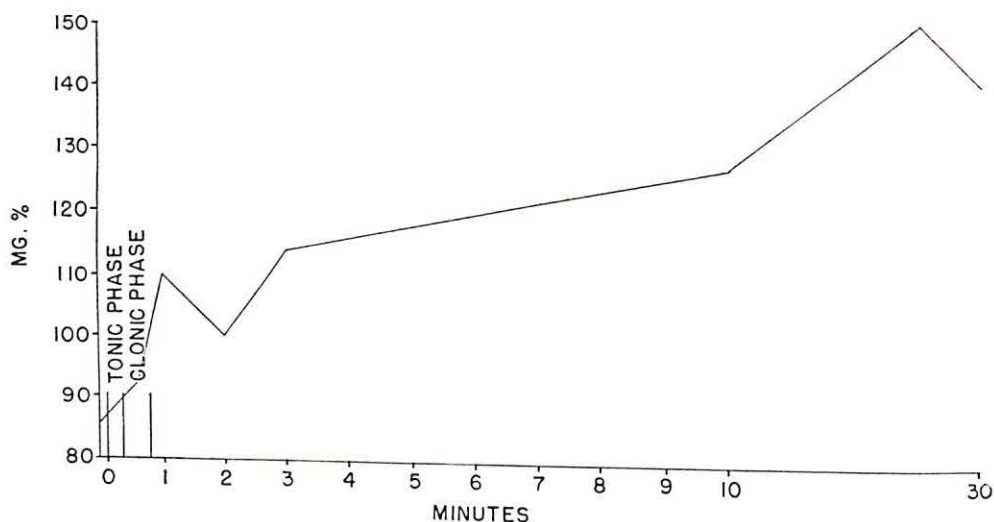


Fig. 34. Glucose in peripheral venous blood. (From the data of Neustadt et al.³⁹)

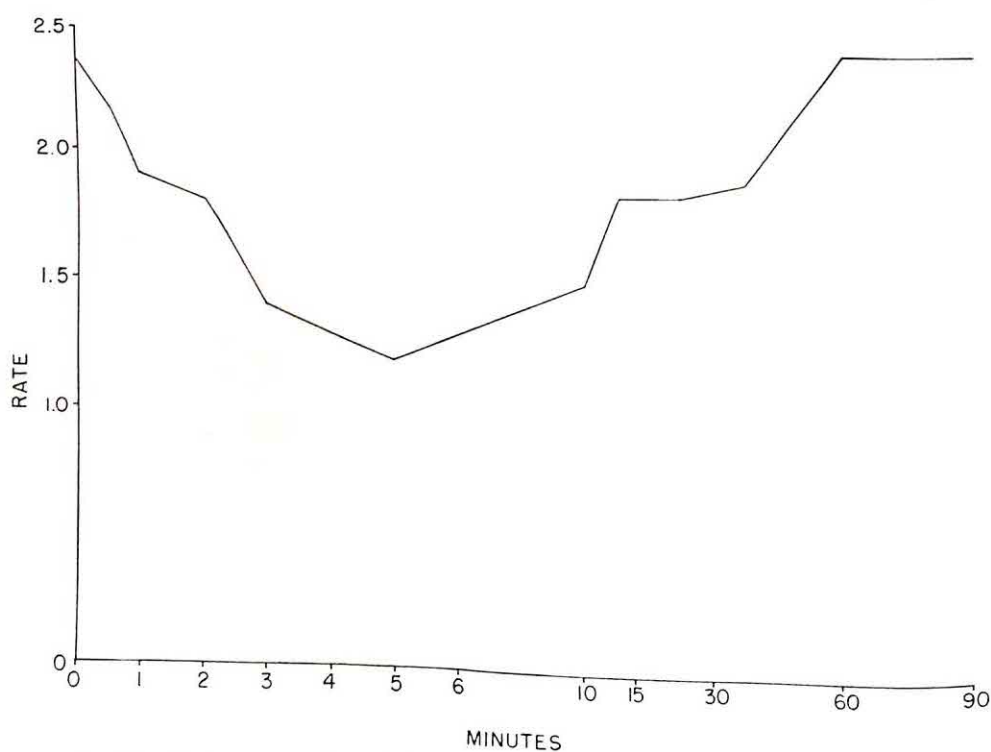


Fig. 35. Cholinesterase in whole blood. (From the data of Neustadt et al.³⁹)

was present during, and for one to one and a half minutes after the convulsion, never outlasting this period. This phenomenon indicated the mobilization of nuclear substances derived from the nervous system during electroshock.

The total protein content underwent significant changes—at first a rise, then a drop, then a sharp rise (Figs. 32 and 36). These variations were caused almost exclusively by variations in the globulin, especially of the internal jugular venous blood. Here, after 3 minutes, a critically low level of globulin sometimes occurred—a fact which may account for otherwise unexplained untoward reactions following electroshock treatment.

An increase in hemoglobin outlasting the convulsion by only 1 to 2 minutes occurred in the peripheral blood exclusively (Fig. 32), unaccompanied by change either in the hematocrit reading or the erythrocyte count. This may be compensatory to the anoxia incidental to the convulsion.

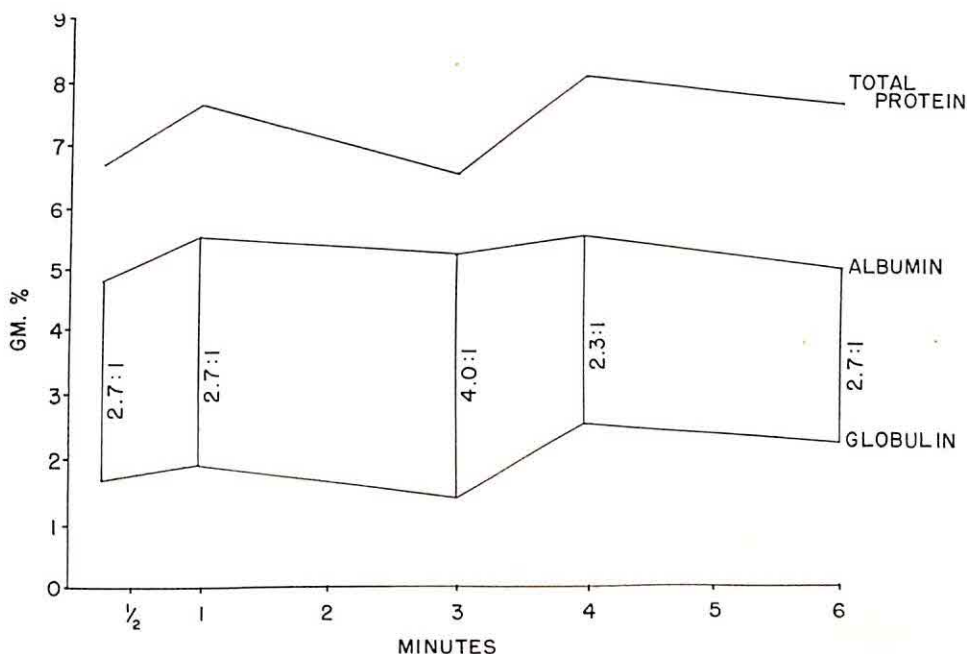


Fig. 36. Protein changes in electroshock therapy. (From the data of Neustadt et al.³⁹)

The total white cell count showed an increase and the differential white cell count a reversal of the figure for polymorphonuclear leukocytes and lymphocytes lasting as long as 8 to 10 minutes (Fig. 32).

Raskin, Howard and Singer⁴⁰ found lymphocytosis and eosinophilia lasting for about 15 minutes after electroshock.

The output of 17-ketosteroids in the urine of our patients (schizophrenics) dropped considerably after five or six treatments, and remained low for six to eight weeks after termination of therapy (Fig. 32). Ashby³³ had found no significant change in 17-ketosteroid excretion in her patients, but a brisk outpouring of other adrenal cortical steroids during the first few days of treatment, without relation to the clinical therapeutic effect of the treatment. Altschule and Parkhurst³⁰ found an increase in the excretion of 17-ketosteroids early in the course of treatment, later a diminution in those patients who showed improvement, whereas no decrease occurred in patients who were not improved. Sackler and his coworkers⁴² confirmed Altschule and Parkhurst's findings. Altschule and his coworkers³¹ later demonstrated an inverse reciprocal relationship in the clinical response of

mental patients to shock treatment on the one hand, and to injection of ACTH on the other. They²⁸ also found indication of increased production of 11-oxysteroids after electrically induced convulsions in man. Stevenson and his coworkers⁴⁴ found in experimental animals that following series of electrically induced convulsions the mean weight of the adrenal glands became increased, while the mean weights of the pituitaries and thyroids of shock-treated animals showed slight but persistent decrease.

Altschule and Tillotson^{29,32} found water retention following shock treatment which they demonstrated not to be due to increased activity of anti-diuretic substances, since the diuretic response to water was accelerated in all patients who gained weight as a result of treatment, whereas it was not accelerated in those whose weight remained unchanged. Welt and his coworkers⁴⁵ confirmed Altschule's and Tillotson's conclusions as to an increase in cellular osmolar concentration following electroshock treatment. Steinfeld⁴³ found relative acidosis following electroshock which he attempted to reproduce by other means with essentially negative therapeutic results. Fetter³⁴ found an increase in gastric acidity following electroshock which he interpreted as due to stimulation of the vagal system brought about by the treatment. This finding is not only of theoretical but also of practical significance, and helpful in the proper understanding and treatment of occasional complaints of heartburn following electroshock therapy.

INSULIN TREATMENT

Insulin treatment consists in the production of a hypoglycemic state sufficiently severe either to reduce or to suspend completely the activity of certain parts of the nervous system. The various parts of the nervous system succumb to hypoglycemia in inverse order of their phylogenetic origin: the most recently acquired and most highly organized parts first and the progressively older ones later.

The mere quantitative reduction of the activity of the cerebral cortex is called *subcoma*. At the moment when cortical activity becomes completely suspended, *coma* begins. The various stages of coma are characterized by successive phases of release and suspension of activity of successively lower layers of the brain stem, a dramatic sequence of events which was first carefully studied and mapped out by von Angyal.^{47,48} Ross and Malzberg,⁷⁴ Frostig,⁵⁹ and Himwich.⁶⁴ It is important to realize, however, that no patient entirely conforms to a precise schema. Some phases may be passed through rather quickly, so that they may almost seem to have been by-passed.

The total time which generally elapses from the phase of decortication (the beginning of coma) to the phase of release of the medulla oblongata (the maximally deep stage of coma that can be tolerated safely, i.e., without serious risk of irreversible brain damage or death) is usually not more than one hour. The time which elapses from the release of the medulla oblongata to the time at which the activity of the medulla oblongata may become suspended is not more than fifteen minutes. It is obvious, therefore, that the limit of safety of the treatment is about one hour of coma, including only fifteen minutes of coma at maximum depth. Since coma sometimes proceeds more precipitously, especially after sensitization to insulin has taken place, it is obvious that coma should be inter-

rupted earlier than after one hour if signs of medullary release have appeared earlier—certainly not later than fifteen minutes after the appearance of such signs. In general it may be said that once there is evidence of suspension of the activities of the midbrain and of the pontine tegmentum, as evidenced by cessation of the light reflex of the pupils and of the response to pain on pressure of the supraorbital nerve, and by abolition of the corneal reflex, it is safe to continue only as long as the pulse remains fast or continues to accelerate slightly, of course provided that there are no alarming signs of circulatory collapse. As soon as the pulse begins to slow during the phase of deepest coma, this is evidence that the dorsal nucleus of the vagus has been released. This event must be taken to predict that a few minutes later the vagus nucleus will become inhibited and irreversible coma or death may ensue. The slowing of the pulse during the final stages of deep coma indicates that the margin of safety has become extremely narrow, and termination of coma by ample amounts of glucose (at least 25, preferably 50 gm., intravenously) must be carried out immediately.

There is no direct and immediate straight line correlation between the various stages of reduction or suspension of brain activity and the mere reduction of blood sugar level. In other words, the blood sugar may drop during the subcoma stage to as low as 35 mg. per 100 cc. and the patient may still be conscious. On the other hand, blood sugar may drop only to 45 mg. per cent during subcoma and may level off at that amount for a half hour, for instance, during which half hour, however, the patient may have lapsed into coma. McGregor and Sandison⁶⁸ found that when they lowered blood sugar rapidly by intravenous injection of insulin to a level as low as 9 mg. per cent within one half hour, patients might still be conscious and alert at that time, lapsing into coma only later. This is due to the fact that reduction or suspension of brain activity is not caused by the immediate effect of the drop in blood sugar but by a gradual process of attrition largely determined by the time over which the brain's most important fuel, namely glucose, has been withheld or supplied in reduced quantities.

Dameshek and Myerson,⁵⁶ as early as 1935, discovered, by means of Myerson's method³⁷ of determination of brain metabolism by observation of the cerebral arteriovenous differences in glucose and oxygen, indicating glucose and oxygen consumption by the brain, that during hypoglycemia the brain metabolism is gradually and increasingly throttled down, the brain gradually consuming less and less of the available glucose as well as of the available oxygen, although the utilization of oxygen becomes generally less severely and less uniformly affected than the utilization of glucose. Himwich⁶⁴ contributed evidence that this discrepancy is probably due to the fact that the brain manages to subsist in small part on its own rather variable glycogen stores during severe hypoglycemia. Loman⁶⁶ made the important discovery that the inability of the brain to metabolize sugar offered in the form of intravenous dextrose outlasts the hypoglycemia and may outlast it for a considerable time depending on the severity of the coma, thus explaining the cause of one of the worst hazards of insulin coma treatment, namely irreversible coma, by lack or impairment of capacity of the brain to utilize sugar following hypoglycemia (Fig. 37).

Insulin produces hypoglycemia by abrogating the brain's priority over

the circulating blood sugar. This priority is normally maintained by the sympatho-adrenal system aided by important synergistic action of the pituitary and thyroid glands, and in women also the ovary. It is particularly the activity of the anterior pituitary which normally, especially in times of stress ("fight-flight"), prevents the absorption and oxidation of glucose by the non-nervous body tissues, while the nervous tissue, par-

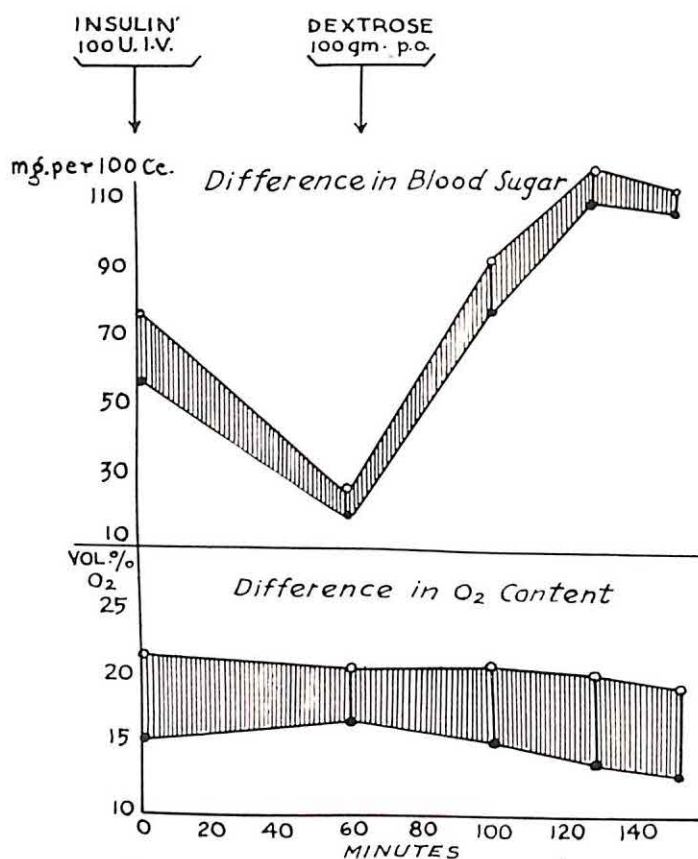


Fig. 37. Arterial-jugular venous differences in blood sugar content (milligrams per 100 cc.) and oxygen content (volumes per cent) during hypoglycemia and during the period of recovery following the administration of dextrose. The curve for arterial blood is represented by a line and hollow circles; that for the blood from the internal jugular vein, by a line and solid circles. (From Loman, Arch. Neurol. & Psychiat., vol. 45, 1941.)

ticularly the brain, remains free to absorb and oxidize sugar at all times, probably because the anterior pituitary secretions are incapable of passing the blood-brain barrier (Himwich⁶⁴). Thus the absorption and oxidation of sugar by the brain goes on independently of the presence or absence of insulin, while the action of insulin—counteracting the inhibition exerted by the anterior pituitary—is necessary to allow non-nervous body tissues to absorb and oxidize sugar. During times of plenty and rest, however, the parasympatho-insulin system takes over, which Cannon^{50,51} has characterized as being that part of the autonomic-endocrine system which serves the function of conservation, protection, and restoration of bodily resources. During such periods, in which the brain is presumed to be at rest, its priority over the circulating blood sugar is abrogated and—probably by

way of inhibition of the anterior pituitary—the non-nervous body tissues are allowed to gorge themselves with glucose and to oxidize it. Part of the energy liberated by its oxidation is utilized to build up glycogen stores, particularly in the muscle and the liver. This process of course is normally allowed to go on only as long as the total blood sugar available does not drop below the sustenance level for the brain. As soon as this takes place, the sympatho-adrenal system takes over again: absorption and oxidation of sugar by the non-nervous body tissues comes to a stop, the brain's priority is restored and the non-nervous tissues again have to subsist on fat and proteins which the brain cannot utilize since brain metabolism is almost entirely dependent on sugar. The fact that the non-nervous tissues subsist in part on sources other than blood sugar is exemplified by the fact that the respiratory quotient of the body tissues is less than 1 (0.7) while that of the brain is close to 1 (0.99).

Injection of a sufficiently large dose of insulin disrupts this homeostatic balance and places the entire organism under the sway of the parasympatho-insulin system. Sugar disappears rapidly from the circulating blood and the respiratory quotient rises toward unity, indicating that all the non-nervous tissues are avidly devouring and rapidly oxidizing glucose which is normally the prerogative of the brain. Since the non-nervous body tissues vastly outweigh the brain, the supply of glucose available to the brain is throttled down and brain metabolism gradually declines, leading—in successive stages—to subcoma and coma respectively.

There are three mechanisms which attempt to maintain brain metabolism during hypoglycemia (Himwich⁶⁴). These are: (1) the formation of glucose from the glycogen stores of the brain itself, (2) the mobilization of glucose from the glycogen stores of the liver, and (3) the utilization of fat by the non-nervous tissues, thus sparing the available carbohydrates for the brain. The latter two mechanisms are sometimes activated by sympatho-adrenal arousal during coma and thus may explain the occasional occurrence of spontaneous awakening from coma especially during the phase of release of the posterior hypothalamus, which will be discussed later. The first two mechanisms probably go on all the time, thus explaining certain discrepancies in the depth of coma achievable by the same dose of insulin in the same patient as dependent upon the available glycogen stores especially of the liver. This explains the frequently observed fact that after a week-end at home the usual coma dose will fail to bring on coma on Monday morning.

The total available glycogen stores are of course limited. Himwich⁶⁴ succeeded in making a fairly accurate appraisal which fits in well with clinical findings and reemphasizes the dangers of allowing hypoglycemia to run through the fifth hour even if coma did not start until the end of the fourth hour. The hepatic quota of 7.5 gm. of glycogen yields approximately 8.3 gm. of glucose; the cerebral quota of 0.8 gm. produces 0.9 gm. of glucose. Assuming that during hypoglycemia the metabolism of the brain is throttled down from a normal consumption of 3.6 gm. per hour to 1.8 gm. per hour, the hepatic and cerebral quota of glycogen would sustain the brain for a total of approximately five hours, a process which the brain could withstand without irreversible damage or death. This calculation clearly highlights the danger of treatments which run into the fifth hour. Chesler and Himwich^{52, 53, 54} have also produced evidence as to the im-

portant causes of the differential susceptibility of the various parts of the brain to hypoglycemia. They have found that the glycogen content—probably representing an indicator of the glucose needs of these parts—ranged as follows: cerebral cortex, 73 mg. per cent; caudate nucleus, 58 mg. per cent; thalamus, 62 mg. per cent; colliculi of the midbrain, 50 mg. per cent; cerebellum, 35 mg. per cent; medulla oblongata, 39 mg. per cent; and spinal cord, 29 mg. per cent, with perhaps 60 mg. per cent as a fair average.

In view of all these complicating factors it is obvious that the physician could not—during the one safe hour at his disposal for coma treatment—be guided in his care for the patient, especially in the proper timing for termination of coma by glucose administration, by the many laboratory procedures which he would need to gain all the pertinent information. The absolute values of blood sugar would tell him nothing. They may be low, for instance 35 mg. per cent, prior to coma; during the early phases of coma they may fall only slightly lower, for instance, to 27 or 20 mg. per cent. On the other hand, during dangerously deep stages of coma the blood sugar, because of arousal of sympatho-adrenal mechanisms, may have risen to 60 mg. per cent without supplying more absorbable glucose to the brain, since the brain's ability to use glucose might be quite severely depressed by that time. Determination of arteriovenous differences would provide the physician with more useful data. However, by the time these determinations could be carried out, it may be too late to utilize them.

The physician must, therefore, in practice rely entirely on his knowledge of clinical neurology and neurophysiology, which must enable him to recognize and interpret correctly the symptoms and signs of the succeeding levels of decerebration that are going on, since he cannot be supplied with really pertinent laboratory data, on which he has increasingly come to rely in other fields of endeavor. If he does not know enough, he may not dare to give comas deep enough to be of therapeutic benefit. He may get scared by a slowing of the pulse during early coma, for instance, when it is merely a symptom of anterior-hypothalamic release and has no portentous significance at all, while during the later stage of coma the slowing of the pulse is of extremely portentous significance. Or his lack of knowledge may make him too foolhardy. He may watch the coma merely by the clock and may fail to recognize the signs of rapidly progressing decerebration in caudal direction. Avoidable deaths not prevented may then make him excessively timid and thereby ineffective subsequently, since in certain patients, particularly paranoids, only comas of maximum depth may be helpful. It is therefore important that the physician have not only theoretical knowledge but practical experience in diagnosing the various depths of coma by means of repeated neurologic examinations and familiarity with the neurologic signs obtainable at the various levels of decerebration, fully realizing, however, that the speed and sequence of the process vary considerably with each patient, as well as in the various phases of treatment. The confusing welter of signs cannot be appreciated by a nurse or by a physician not sufficiently trained in neurology and neurophysiology. It is, however, important to give the nurse some landmarks which she can recognize in the picture, and thus alert the doctor if he happens to be busy with another patient. These simplified landmarks will be discussed more fully in the chapter on the role of the nurse. The physician, however, must

never go by these landmarks alone, but must at all times have a clear appreciation of the coordinated whole of the progress of the patient through the various stages of the coma. Throughout the coma period the doctor must be close at hand and examine the patient repeatedly. In view of their practical clinical importance, I shall give a more detailed account of the neurologic symptoms of the progressive stages of cerebral disorganization during therapeutic hypoglycemia.

Dosages of intramuscular injection of crystalline insulin sufficient to reduce the activity of the cerebral cortex and thus to produce subcoma may vary from 20 units to 160 units. The action of such a dose usually begins one-half hour after injection, and is usually allowed to continue for three hours at which time it is interrupted by permitting the patient to drink a solution of 50 per cent glucose in orange juice. It is wise to give the patient about 1 gm. of glucose for every unit of insulin that has been injected and to have this drink followed by a good breakfast.

It is important to remember that the action of a dose of insulin remains maximal for six hours (Gross,⁶¹ Colwell,⁵⁵ Rohr and Colwell,⁷³ Harrison et al.⁶²), while subsequently its effect gradually declines, retaining significant activity, however, for a total of twelve to eighteen hours, depending on the dose.

Dosages of intramuscular injection of crystalline insulin sufficient to produce complete suspension of cortical activity—thus initiating coma—and then succeeding phases of release followed by reduction and finally suspension of activity of successively lower levels of the neuraxis—thus producing successively deeper stages of coma—usually vary from 100 units to 400 units; larger amounts are necessary in certain cases. If dosages sufficient to produce coma are used, the patient passes first through a preliminary stage of subcoma beginning at about one-half hour after injection and lasting one-quarter of an hour to three and one-half hours before coma supervenes.

(I) The Subcoma Phase. It is preferable to choose a dose which will produce coma after one and a half to two and a half hours of subcoma, that is, two to three hours after the injection of insulin. The first symptoms of subcoma usually appear one-half hour after the injection. The first signs are increased perspiration and salivation, suggesting a release of both sympathetic and parasympathetic activity by a lowering of cortical control. Usually the sympathetic release dominates the symptomatology at first, as it does in the early stages following frontal lobotomy (Rinkel and Greenblatt and their coworkers^{71, 72}). At the same time the patient may feel upset and jittery; fine tremor of the hands makes its appearance and there is gradual increase in tremulousness and incoordination of voluntary purposive movements. After about an hour of this, a phase of relaxation may ensue in which parasympathetic activity gradually comes to the fore. Perspiration will become more profuse and watery, owing to cholinergic release without the simultaneous stimulation of the sebaceous glands. (The latter are sympathetically innervated, thus contributing to perspiration due to sympathetic release a viscous, oily quality.) The pupils will narrow slightly and the patient will drop off to short naps. Whenever the patient becomes stimulated and excited, as by conversation with the examiner, sympathetic activity will again come to the fore, manifested by quickening of the heart rate, slight dilatation of the pupils (Wespi⁷⁵), and viscid quality of the

perspiration. As cortical sedation increases, psychotic content will come increasingly to the fore. If there is depression, the patient may weep and verbalize guilt feelings. Delusional content formerly withheld may be expressed more freely. In catatonic or paranoid patients, psychomotor and other psychotic excitement may be released. However, somnolence and finally clouding of consciousness will gradually supervene. The patient will lose contact with the environment and thus will have lapsed into the next stage, namely, coma.

(II) **Coma.** Coma begins as soon as the patient loses contact with the environment, as ascertainable by his failure to respond in word or activity or other purposive manner to questions, suggestions, and commands on the part of the therapist to which he was found to have responded adequately in the waking state, as well as through the subcoma phase. Asked to turn his head or to show his tongue, he will no longer do so. He will no longer spontaneously address the doctor or answer questions. It is very important to fix the time of onset of coma accurately because, as stated above, the limit of safety of the treatment is almost precisely one hour after onset of coma. Even if the phases of coma proceed more slowly than usual, it is very important to realize that it is unsafe to let even a light coma go on longer than one hour, unless for specific purposes in the hands of a very experienced therapist who must, however, be at all times aware of the danger in which he places his patients and fully convinced of his and his team's ability to cope with it. In general it is well to distinguish three stages of coma, the landmarks of which are important stages in the gradual suspension of cerebral activity at the three major levels: namely, (1) decortication (light coma), (2) decerebration (medium deep coma), and (3) the stage of the hindbrain preparation (deep coma).

(1) *First Stage of Coma: Decortication.* When the patient has lost contact with the environment, his cortex may be presumed to have suspended its activities. The patient no longer asks questions and no longer obeys simple commands. He may, however, still stare at the therapist with wide-open eyes, and may even still follow the therapist across the room with his eyes, without turning his head. This should not be interpreted as a remaining thread of contact with the environment on the cerebral conscious level, such as we must define contact in this connection, because such eye movements in response to visual stimuli may still occur in completely decorticate animals, thereby denoting that they are the expression of a subcortical attention mechanism. I have found this subcortical attention mechanism abolished in the case of a postencephalitic lesion of the midbrain.⁴⁶ Recently Penfield⁶⁹ and Magoun⁶⁷ have investigated this attention-promoting subcortical mechanism in animals and man, and designated it as the "centrencephalic system." It is therefore not surprising that such centrencephalic mechanisms may still be active in the early phases of coma, as long as it remains merely a decorticate state. If this point is not properly appreciated, early phases of actual coma may be misinterpreted as being still merely subcoma. As a result of such a misinterpretation the patient may be allowed to remain in coma for an excessively long time, sometimes to the point of irreversibility of coma.

As decortication goes on, various subphases become distinguishable. Successively lower levels of the neuraxis are first released and subsequently their activity becomes suspended. The earliest release phenomena arise

from the subcortical telencephalic ganglia and the anterior hypothalamus. As the striatum is released, sucking movements of the mouth and other primitive parakinetic movements of the face, trunk and extremities appear, sometimes with athetoid features; forced grasping is common. As the anterior hypothalamus becomes released, perspiration becomes copious and watery, pulse and temperature begin to decline, the pupils may narrow but remain reactive to light. Salivation increases greatly. It is important to change the position of the patient so that the saliva will be able to flow out of his mouth; suction should be available at all times.

Soon the striatal activity subsides and the next lower level, the thalamus, will be released. This is manifested by increased reactivity to sensory stimuli. Any sensory irritation whatsoever evokes abnormally intense reactions. Stimulation of the planta, for instance, will cause vigorous withdrawal movements of the leg and agitated movements of the whole body as if the stimulation were extremely painful. This is obviously a phenomenon of thalamic hyperpathy. Throughout the period of decortication the patient will always correct uncomfortable positions of the body, especially of the neck, thus indicating that the mesencephalic (rubral) righting reflexes function in an unimpaired manner.

Shortly after the release of the thalamus the anterior hypothalamus will go into suspension of activity, and release of the posterior hypothalamus will come to the fore. This manifests itself by a shift from predominantly parasympathetic to predominantly sympathetic release. The pupils will become dilated—still remaining reactive to light—and sometimes exophthalmos will be observed. The pulse rate will accelerate resulting in a flush of the face, and the perspiration will turn viscid. At the height of the sympathomimetic posterior hypothalamic release, outbursts of sham rage may occur either spontaneously or in response to any minor stimulation, such as withdrawal of a blood sample. Sometimes these outbursts of sham rage may be followed by a major epileptic convulsion. If this occurs, the coma should be immediately interrupted. The reason for this is that, because of the anoxia incidental to a major convulsion, the metabolism of the brain becomes so acutely depressed that following such a convulsion the suspension of activity of the posterior levels of the neuraxis may progress so rapidly that within minutes an irreversibly deep stage of hindbrain (medullary) coma with ashen pallor, slow thready pulse, and shallow breathing may ensue. This must by all means be prevented by prompt termination of coma immediately after or preferably still during the epileptic fit.

The release of the posterior hypothalamus with its sham rage and lowered seizure threshold is almost always a stormy process in paranoid and in catatonic patients. There are other cases, however, in which the phase of release of the posterior hypothalamus appears to be by-passed in that neither excitement nor sympathetic release ensues at this stage, the patient sliding almost unnoticed into the next stage, that of decerebration. This by-passing of release of the posterior hypothalamus is particularly characteristic of cases of simple schizophrenia and of patients with obsessive-compulsive states. It almost appears that in these types of patients the posterior hypothalamic mechanisms are either insufficiently strong, and thereby not susceptible to adequate release, or so sensitive that they are immediately inhibited together with the more rostral parts of the hypo-

thalamus. Adrenal mechanisms cannot be held responsible for this variation, since the work of Gellhorn, Cortell, and Feldman⁶⁰ in animals has shown that the rage reaction itself was unaffected by denervation of the adrenals or sectioning of the spinal cord. These authors found, however, that the associated sympathomimetic excitation merely predominates in the intact animal, but that an associated lesser degree of parasympathetic excitation can be demonstrated after denervation of the adrenal glands or sectioning of the spinal cord.

This particular phenomenon of "by-passing" of certain stages of release emphasizes the great individual variations in the progress of the levels of coma. The physician cannot count on any particular phenomenon to appear as a landmark, but at all times must be ready to recognize successively lower landmarks as soon as they appear in the clinical picture.

The decorticate phase of coma with its stages of striatal, anterior hypothalamic, thalamic, and posterior hypothalamic release and subsequent suspension may last from twenty minutes to one hour, in most cases about one-half hour. If this decorticate phase ("light coma") alone has lasted a full hour, the coma should be interrupted and no deeper phases of coma be allowed, unless an experienced therapist, after thorough acquaintance with the patient's reactions and responsiveness to resuscitating measures, has found it both safe and beneficial to allow slightly longer comas. The physician should, however, increase the next day's dose, in order to accelerate the phasic progression of the coma to be expected if deeper stages of coma are desirable therapeutically. This will have to be the case particularly with paranoid patients whose response to treatment is best with comas of maximum depth.

(2) *Second Stage of Coma: Decerebration (Medium Deep Coma)*. As soon as the posterior hypothalamus and the red nuclei become inhibited, the next phase of coma begins, namely, decerebration. This stage is characterized essentially by a mesencephalic release posterior to the red nuclei, the red nuclei appearing to be released and inhibited together with the posterior hypothalamus rather than with the remainder of the midbrain.

The first sign that this phase has begun is the loss of the righting reflexes. The patient will no longer correct uncomfortable positions of the extremities and particularly of the head, and he will be much less responsive to external stimuli in general, although pressure on the supraorbital nerve will still produce withdrawal and grimacing. Parasympathetic-cholinergic activity will again come to the fore, the sweat turning watery and the pupils contracting. Tonic spasms with arms flexed and legs extended may appear, as well as slow torsion movements, although some patients may be limp and quiet. The sign of Babinski will be unequivocally positive in this phase, while prior to this phase it may be obscured by the withdrawal reflex. As long as the midbrain is released, the pupils will remain reactive to light, since the light reflex of the pupils is transmitted through the anterior colliculi of the midbrain; the patient will also react to pressure on the supraorbital nerve because this type of deep pain reflex is transmitted not through the thalamus but through the posterior colliculi as well as the tegmentum of the midbrain (Foerster⁵⁸). Close supervision of the patient is necessary throughout the mesencephalic phase of decerebration because the patient may at any time, not only after the customary ten to twenty

minutes, slide into the next deeper phase, namely, the phase of release of the hindbrain, the safety limit of which is only fifteen minutes.

(3) *Third Stage of Coma: The Stage of the Hindbrain Preparation (Deep Coma)*. This stage must be presumed to have begun as soon as important midbrain activities are found to have become suspended; foremost among these are the light reaction of the pupils and the wincing or turning reaction to pressure on the supraorbital nerve. As soon as the anterior part of the hindbrain is released, sympathetic activity again comes to the fore. The sweat turns viscous again, the pupils dilate, but are no longer reactive to light. The pulse accelerates. Tonic spasms with extension of arms and legs may appear. These spastic phenomena are now entirely of the extensor type, obviously due to the release of the tonus-regulating nuclei in the reticular substance of the hindbrain, both of the pontine tegmentum and of the medulla oblongata. Attacks of such extensor spasms may become particularly prominent whenever respiration becomes labored or obstructed owing to accumulation of mucus. Suction apparatus should therefore be available and resorted to whenever necessary. During this hindbrain phase of coma, the patient is safe as long as the pulse rate accelerates or stays fast—but below 140 beats per minute. Any decline of the pulse rate during this phase heralds the approach of its safety limits because such a decline means that the dorsal nucleus of the vagus located at the caudal extremity of the medulla oblongata—by nature the most resistant to hypoglycemia of all the parts of the brain stem—is being released, which is the last phase before its activities would become impaired and finally suspended. Suspension of the activities of the vagus, however, is incompatible with life, and the approach of this danger means the risk of fatal irreversible coma. The physician, therefore, must take great pains to prevent such a life-threatening emergency from arising by interrupting deep coma before its safety limit is reached. He must interrupt the hypoglycemia after a maximum of fifteen minutes of deep (hindbrain) coma, or prior to that, whenever the pulse rate slows down during this phase of coma.

Another important landmark in this deep phase of coma is the abolition of the corneal reflex. When this takes place it means that the trigeminal or the facial nuclei or both have become inhibited. These nuclei are situated within the border area where the anterior hindbrain (the pontine tegmentum) and the posterior hindbrain (the medulla oblongata) are joined together, and are located just a few millimeters forward from the vagus nuclei. The fact that the corneal reflex has been abolished means that the activity of the anterior part of the hindbrain, the metencephalon, including the pontine tegmentum, has been suspended, and that the only part of the brain stem that is still continuing to function and thus to maintain life is the posterior part of the hindbrain, the myelencephalon or medulla oblongata. As long as the nuclei of the reticular substance of the anterior part of the medulla oblongata are active, the pulse will remain fast or accelerate further, the nonreacting pupils will remain moderately dilated, but the skin will become increasingly pale and the body temperature continue to fall. As soon as the activity of the anterior medulla oblongata declines and the posterior medulla oblongata, including the vagus nucleus, is released, there is a definite shift back to parasympathetic activity. The nonreacting pupils become narrow; sweat becomes watery;

body temperature falls abruptly; the pulse rate declines and respiration becomes shallow. All life processes become slowed down to a minimum. It is up to the therapist to see that this cerebral depression does not go to a point not compatible with complete recovery. It is best to interrupt deep coma at the time when the corneal reflex becomes abolished. Such comas will have been deep enough for the therapeutic effect even in those conditions which require treatment by maximally deep comas, and the final and dangerous phase of vagal release with its marked depression of circulation will have been safely avoided.

It is the marked depression of circulation and respiration during the final phase of vagal release which subjects the patient to the greatest risk of irreversible brain damage, quite apart from the dangers of irreversible coma and death.

Interruption of Hypoglycemia. If interruption of hypoglycemia is carried out in time, resuscitation will be prompt. Interruption can be carried out either intravenously or by gastric gavage. Intravenous administration of 8 to 25 gm. of glucose (I prefer to give 25 gm. in the form of 100 cc. of 25 per cent dextrose after every deep coma, irrespective of the magnitude of the insulin dose) will resuscitate the patient within five minutes. At that time, additional glucose must be given by mouth: a total of 100 gm. of glucose for every 100 units of the original dose of insulin will generally suffice to prevent the patient from slipping again into a delayed coma during the subsequent afternoon, although not necessarily during the subsequent night. This particular danger will be dealt with, together with measures for its prevention, in a subsequent chapter.

Gastric gavage by nasal tube of an appropriate amount of 50 per cent glucose (100 gm. for every 100 units of insulin is generally sufficient) should resuscitate the patient within fifteen to twenty minutes.

If resuscitation has not taken place either within five minutes after the original intravenous interruption or within twenty minutes after the original gastric interruption, an additional dose of 25 gm. of glucose should be administered, this time always by vein. If the patient fails to be resuscitated within five minutes after this additional dose, the diagnosis of irreversible coma must be made immediately and the full regimen for the relief of irreversible coma must swing into action. This regimen includes continuous intravenous infusion, oxygen, and other emergency measures which will be fully described in Chapter X, dealing with complications and lifesaving measures.

It is important to realize that the time schedule of the progress of coma is a variable one and the physician must at all times be ready to cope with unexpected developments. He must also realize that from day to day, shifts in the susceptibility to insulin may become apparent, which von Braunschweig⁴⁹ has ascribed to "sensitization."

I should like to give a practical example (Fig. 38) in the case of the 58th insulin coma treatment of a 33 year old paranoid schizophrenic male patient. This patient gradually went into his final comas faster as treatments progressed. Since maximally deep comas were desired it was felt unwise to reduce the dose at that point, and preferable to take particular pains in the precise management of the comas. This case also illustrates the important fact that there is no direct immediate relationship between blood sugar level and depth of coma.

Prior to the treatment the patient's blood sugar (fasting) was 116 mg. per 100 cc. (Fig. 38). The patient was then given 160 units of insulin intramuscularly. Half an hour later his blood sugar had fallen to 45 mg. per cent and

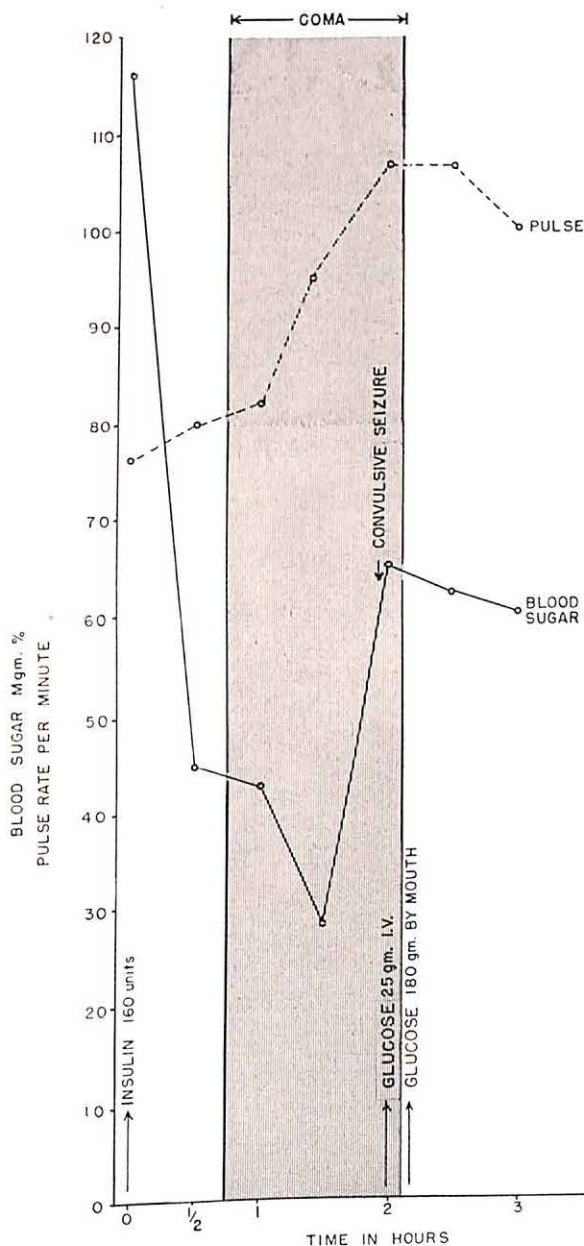


Fig. 38. Blood sugar curve and pulse curve before, during, and after insulin coma treatment. The period of coma is indicated by the shaded area of the graph.

he began to perspire slightly, but he was still awake, reading, pleasant, and cooperative. Fifteen minutes later his speech became thick; slight twitching of face and body became noticeable; perspiration became profuse and watery. There was motor restlessness and the patient slipped out of contact. Coma was therefore found to have begun then, that is, 45 minutes after the injection of

insulin. The blood sugar, however, showed no further significant decrease when tested 15 minutes later; it was still 43 mg. per cent, although the patient by that time was definitely well in the decorticate phase of coma. It is important to realize that the patient had still been awake at a practically identical level, namely 45 mg. per cent, only one-half hour before.

Half an hour later, 45 minutes after onset of coma, and $1\frac{1}{2}$ hours after the injection of insulin, blood sugar had declined to 28 mg. per cent. At the same time the patient's pulse, which had not changed significantly during the first hour after injection, had increased from 76 to 95 beats per minute. At this time the patient's coma had progressed to the phase of thalamic and posterior hypothalamic release; this phase is still part of the first stage, i.e., of light or "decorticate" coma. The patient showed motor restlessness with sham rage and loud ululations. There was marked irritability to slight sensory stimuli, and the patient promptly corrected uncomfortable postures. There were no other purposive actions or reactions. Sweat was turning sticky, indicating sympathetic release. Attempts to examine his pupils elicited outbursts of sham rage. This condition was still present 15 minutes later when the patient had spent one full hour in coma. In view of the desirability of maximally deep comas in this paranoid patient, and because his safe tolerance of deep coma had been found greater than average during his preceding 57 coma treatments, it was decided to go on with coma, watchfully hoping that he could be allowed to slide into the mesencephalic phase of decerebration. Fifteen minutes later, however, in conjunction with one of his outbursts of sham rage, a major grand mal convulsion was elicited. Five minutes after the convulsion the patient's blood sugar was found to have risen to 65 mg. per cent, without any alteration in his state of coma. The treatment was then interrupted by injecting 25 gm. of dextrose in 100 cc. of water intravenously. Recovery was delayed, requiring 10 minutes, after which time an additional 180 gm. of glucose was given by mouth in the form of a 50 per cent solution. The fact that resuscitation following intravenous interruption of hypoglycemia was delayed beyond the usual time of 5 minutes indicated that the safety limit had indeed been reached shortly after the convulsion, although the patient was only in light coma prior to the convulsion and in spite of the fact that the blood sugar had risen to 65 mg. per cent as a consequence of the convulsion and the preceding phase of sympathetic release.

Half an hour after termination of treatment, the patient was out of coma, complaining only of a slight headache; his blood sugar, however, had declined slightly further, namely, to 62 mg. per cent. Another half hour later his blood sugar had slightly fallen still further, namely, to 60 mg. per cent, but the patient was fully conscious, pleasant, cooperative, and about to consume an appetizing meal.

ETHER DRIP INFUSION TREATMENT

Ether drip infusion treatments were first introduced by Katz⁷⁸ and Williams⁷⁹ for the treatment of states of impaired circulation, including cases of impending gangrene. The rationale of the treatment was its relaxing effect on blood vessel walls which appeared to counteract sympathetic stimulation. Since enhanced sympathetic tone is a significant psychosomatic characteristic of depression, the use of this measure in depressions and other affective psychoses appeared promising; it was first introduced for this purpose by Ferraro⁷⁶ and his coworkers⁷⁷ in 1950.

The treatment consists in the intravenous administration of a $2\frac{1}{2}$ per cent solution of ether in 1000 cc. of 5 per cent dextrose in isotonic sodium chloride solution. It is given by slow phlebotomy, starting with about 30

drops per minute and allowing infusion gradually to accelerate to 180 drops per minute. Total treatment time for the administration of 1000 cc. is usually from 2½ to 3 hours. Treatments are given daily. Ferraro and his coworkers⁷⁷ gave from 10 to 27 treatments in each series of daily treatments.

Slow intravenous ether drip infusion does not produce narcosis or even partial interference with the patient's consciousness. The treatment has a pleasant, relaxing, only slightly sedative effect in which significant signs of cholinergic stimulation come to the fore. There is increased diaphoresis, congestion of the face and conjunctivae, as well as increased diuresis. The pulse rate decreases in some patients and does not show significant change in others. In those patients in whom the blood pressure was elevated because of enhanced sympathetic tension prior to treatment, the blood pres-

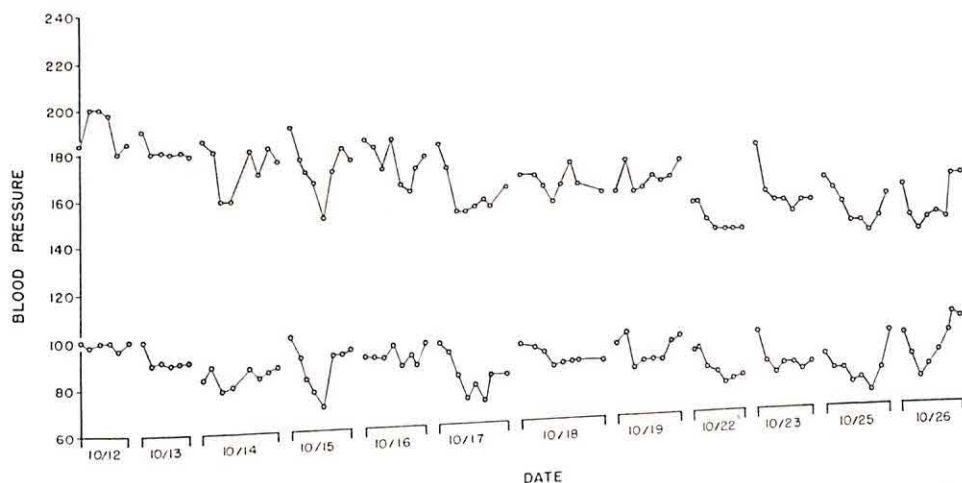


Fig. 39. Systolic and diastolic blood pressures during twelve consecutive ether drip infusion treatments lasting 2½ to 4 hours each. Note tendency of blood pressure to drop usually about halfway through each treatment, as well as to decline gradually throughout the two weeks of treatment. Female, aged 57 years. Chronic depression of seven years' duration. The treatment brought about temporary social recovery lasting one year.

sure dropped during treatment, reaching its lowest point usually halfway through each treatment; in addition there was also a gradual lowering of the blood pressure from day to day as the treatment series progressed, the basic blood pressure being significantly lowered at the end of a successful treatment series (Fig. 39).

In summary, it may be said that the treatment consists in a strong cholinergic stimulus which appears to counteract the enhanced tone of the sympathetic system characteristic of depressive states.

FRONTAL LOBOTOMY AND RELATED PSYCHOSURGICAL PROCEDURES

Undercutting, Topectomy, Thalamotomy and Temporal Lobe Surgery
(temporal lobectomy, temporal lobotomy, amygdaloidectomy)

The relief of anxiety, hostility, obsessions, compulsions, paranoid projections, depression, and of those aspects of pain which produce threat, panic, and persistent obsessive emotional suffering, which can be brought about by successful frontal lobotomy is probably induced by interruption

of the connections between the frontal lobes and the thalamus. Recent research indicates that not all these connections are necessarily direct ones, although interruption of the direct pathway, namely, the anterior peduncle of the thalamus, was a most striking finding obvious to the early investigators of the problem. Freeman and Watts⁹² found that the anterior peduncle of the thalamus is largely a thalamofrontal tract, since the retrograde degenerations of the ganglion cells of origin are predominantly thalamic rather than cortical. Cortical degeneration especially of certain parts of the granular layers is certainly far less conspicuous and extensive than the retrograde degenerations of cells in the thalamus. Freeman and Watts⁹¹

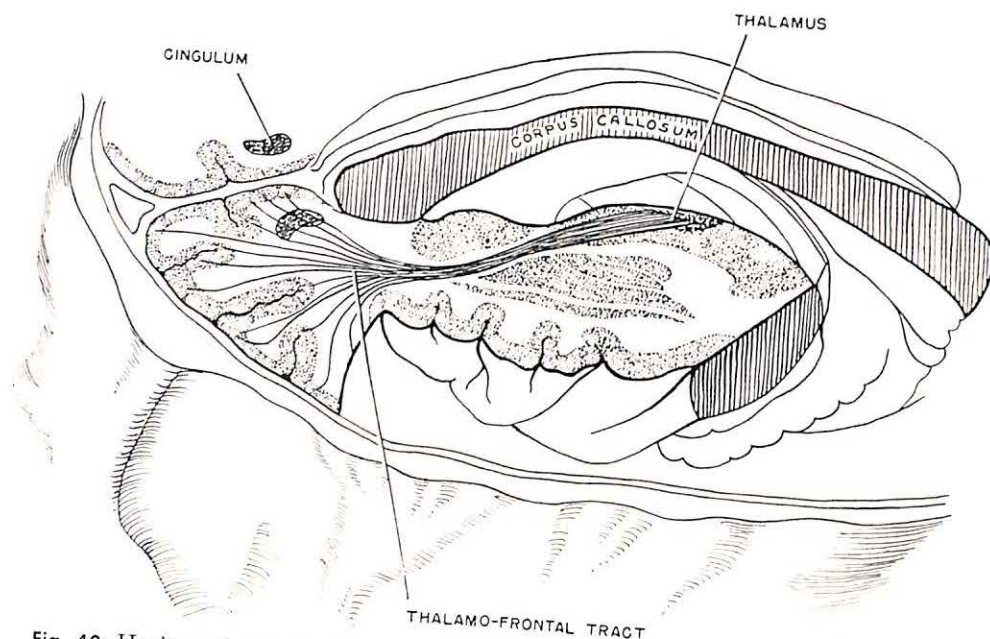


Fig. 40. Horizontal section through the thalamofrontal tract in its topographic relationship to the corpus callosum and the landmarks of the skull (with partial utilization of an illustration from Zuckerkandl: *Atlas der topographischen Anatomie des Menschen*).

found essentially that the medial nucleus of the thalamus projects to the frontal pole, the anterior nucleus to the limbic lobe (the cingular gyrus), and the lateral nuclear group to the lateral convexity of the frontal lobe. In studying cases with up to ten years' postoperative survival they found that retrograde degeneration of the small-celled dorsal part of the medial nucleus of the thalamus is the most consistent correlate with good clinical results of the operation. Spiegel and Wycis and their collaborators,¹⁴⁵⁻¹⁵⁰ proceeding on the basis of these findings, destroyed the dorsomedial nucleus of the thalamus by means of a needle electrode inserted into the thalamus under the guide of a stereotaxic instrument, a modified Horsley-Clarke apparatus. They reported that destruction of as little as one-seventh of that nucleus by electric coagulation produced favorable clinical results comparable to those of frontal lobotomy. Thus the prominence of the thalamofrontal system in achieving the clinical results of frontal lobotomy seemed established.

Meyer, Beck, and McLardy,^{112-123, 85, 86, 87} as well as Yakovlev, Hamlin,

and Sweet,¹⁵⁵ however, pointed out that in most successful cases of frontal lobotomy other fiber systems are destroyed as well, notably the cingulum and the tractus uncinatus, which have no direct connections with the thalamus. Meyer and Beck¹¹⁷ contributed one important successful case of frontal lobotomy in which the surgeon had merely incised the insula and the opercular region of the frontal lobe, probably including the tractus uncinatus. In this case degeneration of the medial nucleus of the thalamus was entirely absent. This case confounded the conceptual scheme of frontal lobotomy as essentially an interruption of the direct thalamofrontal radia-

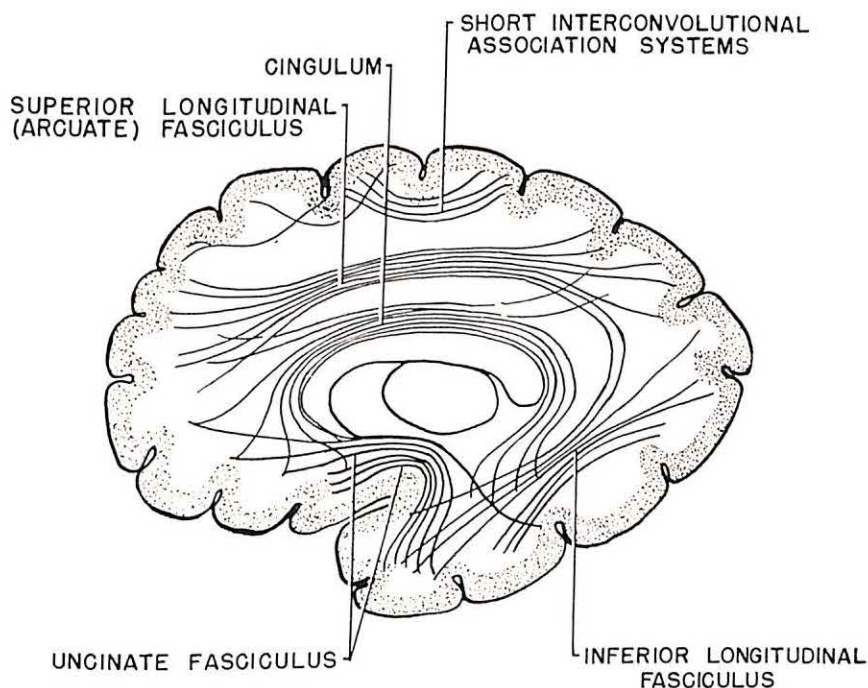


Fig. 41. The major association systems of the cerebral hemisphere (after Meynert).

tion, especially in view of the fact that this patient who survived the operation for 16 months had achieved a fairly good clinical result.

In order to understand the complexity of the problems involved, it will be best at this point to review the projection and association systems which unite the frontal lobes with other parts of the brain (Figs. 40-44).

(1) *The Thalamofrontal Radiation.* If we trace this radiation backward from the frontal lobe in a horizontal section (Fig. 40), we find that the fibers converge backward toward the putamino-caudate narrows of the anterior capsule which contains largely the thalamofrontal radiation as well as the frontopontine tract. The fibers derived from the medial section of the frontal lobe lie adjacent to the caudate nucleus; those from the lateral part of the frontal lobe lie adjacent to the putamen. Prior to entering the putamino-caudate narrows, these fibers skirt the lateral aspect of the anterior horn of the lateral ventricle.

(2) *The Fasciculus Longitudinalis Superior of Burdach.* This large association system connects the various lobes of the brain from the frontal

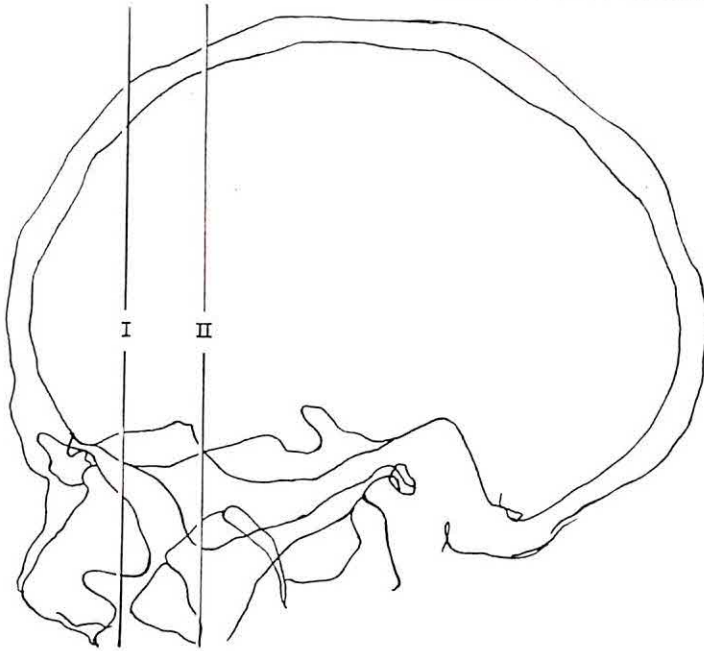


Fig. 42. The two most frequently employed levels of frontal lobotomy in their relationship to the skull.

I. Anterior type of lobotomy. The fiber tracts sectioned at this level are illustrated in the transverse section shown in Fig. 43.

II. Posterior type of lobotomy; the section passes a few millimeters behind the sphenoid ridge. The fiber tracts sectioned at this level are shown in transverse section in Fig. 44. (Outline adapted from Schüller and Urban: *Craniocerebrale Schemata für die roentgenographische Lokalisation.*)

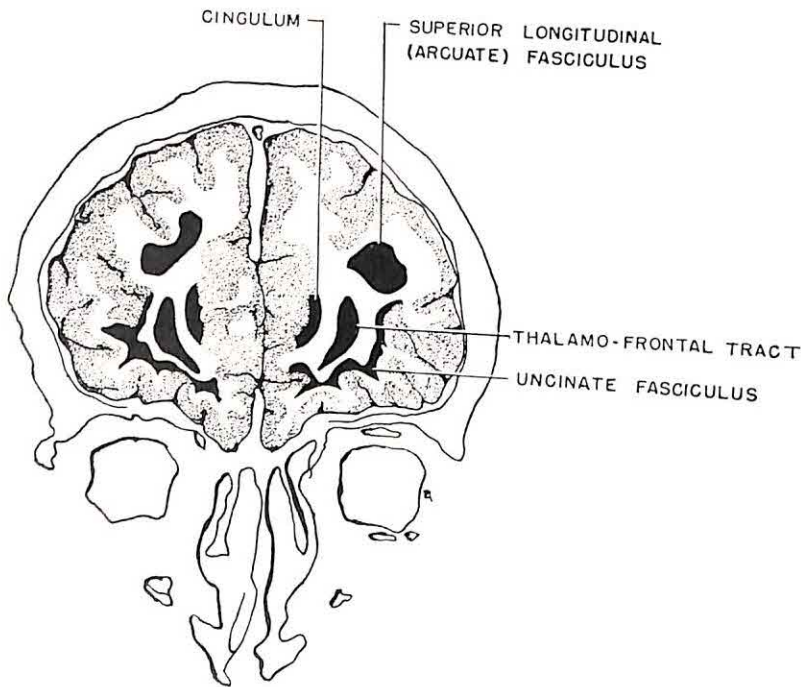


Fig. 43. The major cortical association systems (in black) at the level of an anteriorly placed frontal lobotomy. The outlines of the brain and the landmarks of the skull are adapted from Schüller and Urban.¹⁴²

to the occipital lobes (Fig. 41). It lies dorsal and lateral to the usual area involved by frontal lobotomy (Figs. 43 and 44) and was found by Meyer, Beck, and McLardy as well as by Yakovlev, Hamlin, and Sweet to be essentially spared in successful lobotomies.

(3) *The Cingulum (Burdach)*. This important association system surrounds the corpus callosum anteriorly, above, and posteriorly (Fig. 41), lying in close connection with the gyrus cinguli (Figs. 43 and 44). It con-

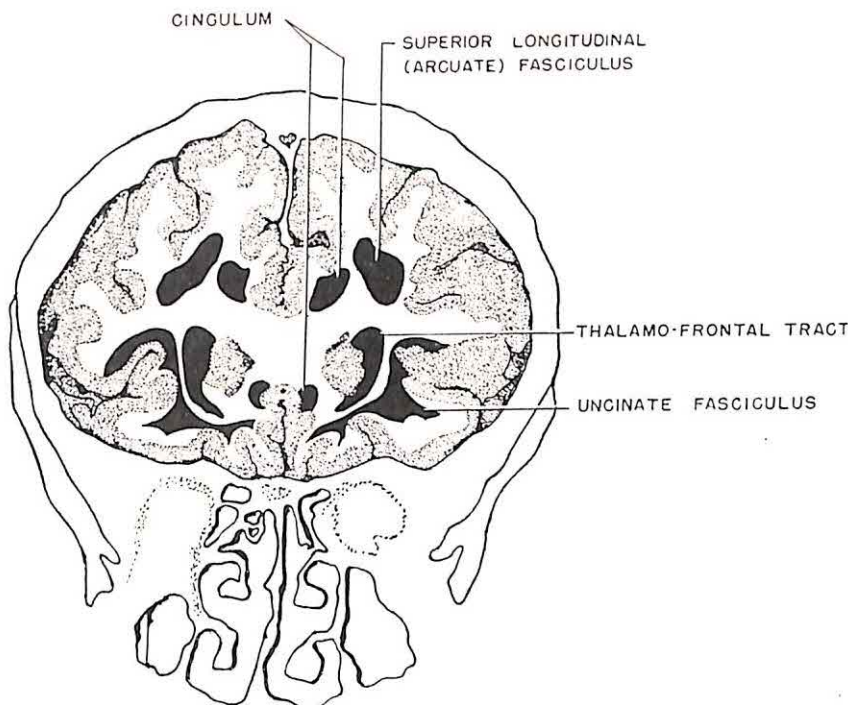


Fig. 44. The major cortical association systems (in black) at the level of a posteriorly placed frontal lobotomy. The outlines of the brain and the landmarks of the skull are adapted from Schüller and Urban.¹⁴²

nects the cingular lobe or limbic cortex with the temporal lobe, including the uncus of the gyrus hippocampi, sending a small bundle to the parietal lobe.

(4) *The Fasciculus Subcallosus of Forel-Onufrowicz-Kaufmann* is largely a fronto-occipital bundle. It lies immediately beneath and laterally to the corpus callosum and takes part in forming the tapetum, i.e., the upper mesial wall of the lateral ventricles. This bundle was found to be degenerated following successful frontal lobotomy by Yakovlev and his co-workers.¹⁵⁵

(5) *The Fasciculus Longitudinalis Inferior of Arnold*. This bundle connects the occipital with the temporal lobes (Fig. 41), according to Flechsig also with the thalamus, forming part of the posterior peduncle of the thalamus. This bundle is not affected by frontal lobotomy. (The temporal lobe is in connection with the thalamus through the lateral peduncle of the thalamus.)

(6) *The Fasciculus Uncinatus*. This bundle extends from the orbital

part of the frontal lobe past the insular region to the temporal lobe (Figs. 41, 43, 44); there it also gives important fibers to the gyrus hippocampi and the amygdalar complex as well as sending fibers to the cingulo-limbic cortex during the proximal part of its course. This bundle is degenerated in many successful cases of frontal lobotomy.

(7) *The Fasciculus Occipitalis Verticalis of Wernicke*, which connects the fusiform gyrus with other parts of the occipital and the posterior parietal area. This bundle, of course, is not affected by frontal lobotomy.

(8) *Broca's Bundle* extends from the tip of the hippocampal gyrus through the substantia perforata anterior to the gyrus cinguli. This bundle may become affected in posteriorly placed frontal lobotomies, although no specific report of its being affected has been made.

(9) *The Fornix*. The fornix is a projection system arising from the temporal lobe, particularly its hippocampal and amygdaloid complex, swinging backward beneath the splenium of the corpus callosum, then again forward close to the substantia perforata anterior and then passing backward beneath the lateral wall of the third ventricle to end in the mammillary body.

From the mammillary body a new bundle arises named Vicq d'Azyr's bundle which goes forward again within the lateral wall of the third ventricle in the paramedian part of the thalamus, to end in the anterior nucleus of the thalamus. From the anterior nucleus of the thalamus the next relay of fibers passes backward in the habenula along the tenia thalami to end in the ganglion habenulae at the posterior extremity of the third ventricle; there the ganglion habenulae sends important fibers to the pineal gland. From the ganglion habenulae the next main relay of its emerging fibers extends downward in the form of Meynert's fasciculus retroflexus to the base of the brain, namely, to the space between the two cerebral peduncles; there they end in the interpeduncular ganglion. From the interpeduncular ganglion the next neuronal extension goes upwards again in the form of raphe fibers through the foot region of the midbrain, to end in Gudden's nucleus which lies at the base of the sylvian aqueduct behind the trochlear nucleus. From there the next chain of neurons arises in the form of fine, scantily myelinated fibers which pass through the periaqueductal gray of the isthmus region and beneath the floor of the fourth ventricle where they form the so-called dorsal longitudinal bundle of Schuetz. These fibers terminate close to the nucleus of the vagus nerve in the nucleus intercalatus of Staderini, where they are joined by fibers from the vestibular nuclei which enter the nucleus intercalatus by means of Fuse's bundle. From the nucleus intercalatus a short tract of neurons sets out which ends in the dorsal or cardio-pneumo-gastric nucleus of the vagus.

Thus, it can be seen that these impulses arising from the temporal lobe, as well as similar ones from the frontal areas, and those as well, of course, which have come from the frontal areas to the temporal lobe by means of the uncinate bundle, finally reach the vegetative nuclei of the hindbrain, especially those of the vagus nerve. This highlights the direct participation of cortical impulses in modifying the lower motor neurons of the vegetative system, a physiologic fact pointed out by Fulton.⁹⁶ It is important to realize that the cortex has such close connections with the vegetative system because clinicians all too often ascribe abnormal neurovegetative phenomena primarily to the hypothalamic vegetative nuclear systems which

are certainly in part by-passed by the fornical system just described. Fulton^{96a} very aptly states: "These new disclosures concerning the relation of the cerebral cortex to the autonomic system give an adequate physiologic basis for the relationship long recognized between mental states and vegetative processes . . . the heart and circulation may be worked just as hard and just as much to the detriment of the body as a whole from an arm-chair—or perhaps I should say a swivel chair—as from a rower's seat. Many disturbances of gastro-intestinal function undoubtedly have a similar basis."

It is important to realize that in addition to the direct thalamofrontal radiation connections also exist which connect the thalamus by means of the temporal lobe with the frontal lobe. This explains a good many otherwise inexplicably complicated responses following frontal lobotomy. Meyer and Beck's¹¹⁷ famous case can certainly be explained by the fact that the uncinate tract which connects the frontal lobe with the temporal lobe and through it indirectly with the thalamus was sectioned. Williams and Freeman¹⁵⁴ in a recent paper have aptly compared this dual connection of the thalamus with the frontal lobe to the pyramidal system and the extrapyramidal system respectively. Like the pyramidal system, the thalamofrontal system directly connects a higher cortical center with a center of a lower order, while the thalamo-temporo-frontal system forms an indirect relay system comparable to the arrangement found in the extrapyramidal system. As in the extrapyramidal system, one of the centers implicated in this indirect relay is an older derivative of the telencephalic vesicle, namely the amygdaloid nucleus. Thus the amygdaloid nucleus is comparable to the striatum, which also is an old paleocephalic derivative of the telencephalic vesicle. Here also a lower center, namely the thalamus, which is part of the diencephalon, is connected with a higher cortical center, namely the frontal lobe, through a relay station composed of more ancient derivatives of the telencephalic vesicle, in this case the amygdaloid nucleus and the associated allocortical or mesocortical parts of the temporal lobe.

Effects of Operation. The results of frontal lobotomy are immediate as well as slowly progressive. The period of convalescence during which further improvement can be expected may last two or more years.

Freeman and Watts⁹³ early in their work started using local anesthesia so that the patient could report his feelings and ideas during the course of the operation as well as respond to psychologic tests as guideposts to the process of lobotomy. They observed a dramatic alteration in mental state when the basomedial part of the last quadrant was incised regardless of the order of incising the other three quadrants. Freeman and Watts^{92, 93} used the word quadrant to describe the upper and lower half of each frontal hemisphere; visualizing both frontal hemispheres as a circle, each upper and each lower half comprised one quadrant according to their terminology. Even with both upper and one lower quadrant incised they found that the conscious patient was still oriented and still capable of reporting a degree of anxiety and distress almost as great as in the preoperative state, but "in a matter of seconds after incision of the fourth quadrant the patient becomes unresponsive, disoriented, confused and is freed completely from his anxieties." Pulse and blood pressure fall, flushing and sweating occur, and some patients appear to go to sleep. Many patients, while still on the

table, following incision of the last quadrant are unable to recall their immediate surroundings and may even deny the operation. Freeman and Watts⁹² observed this disorientation so frequently that they came to regard it as a characteristic necessary for the success of the operation.

The period of postoperative convalescence may vary from a few weeks to as much as one year; however, as has been pointed out, a number of additional years may still be classifiable as a period of convalescence in that further improvement accrues, possibly by reestablishment of frontal activity transmitted by the remaining intact association systems, especially the arcuate fasciculus. Most convalescences are marked by a period of incontinence which may vary from a few days to a month or longer and a certain degree of dullness and slowing which is transitory in successful cases. The incidence of convulsions will be taken up in the next chapter dealing with complications.

Bilateral frontal lobotomy is always necessary for the relief of mental disease in contrast to the relief of pain in which Scarff¹⁴¹ and Koskoff and his coworkers¹⁰⁶ succeeded in producing relief by unilateral operations. In mental disease, however, unilateral operations are ineffective even if the better vascularized and hence presumably more active frontal lobe is operated on first. Dr. James L. Poppen¹³³ carried out this procedure for a time in order to avoid excessive depletion following lobotomy. In a patient of mine who suffered from a known unilateral cerebral atrophy (probably Bielschowsky's disease), the fact that the left hemisphere was atrophic was confirmed by pneumoencephalogram and electroencephalogram, as well as by arteriogram. Frontal lobotomy carried out on the right normal side produced no change in the patient's mental picture. A week later, however, when lobotomy was carried out on the left side, relief was instantaneous and has progressed to a state of adequate social recovery from the mental illness. Inspection of the left side of the brain at operation confirmed the finding of atrophy which had been indicated by pneumoencephalographic, electroencephalographic, and arteriographic examinations. When Dr. Poppen incised the white matter of the left atrophic hemisphere he found that the white matter was more gray in color, was of softer consistency and showed fewer bleeding points than the equivalent area on the right.

The neurovegetative changes following frontal lobotomy in man essentially confirmed the findings made earlier in animal experiments. As early as 1894 Spencer¹⁴⁴ found that stimulation of the orbital surface in dogs, cats, rabbits, and monkeys caused arrest of respiratory movements as well as changes in the blood pressure. With stimulation in the cingulate area some foci produced elevations of blood pressure, others a fall. In 1938 Bailey and Bremer⁸¹ observed that stimulation of the central cut end of the vagus nerve in the cat's isolated head reflected itself in the electroencephalogram of the posterior part of the orbital lobe in what later was designated as Walker's area 13, and in no other part of the cerebral cortex. Bailey and Sweet⁸² found that stimulation of this same region caused respiratory arrest and blood pressure changes similar to those described by Spencer. Chapman and R. and K. Livingston¹⁰⁸ essentially confirmed these findings for man, finding arrest or lowering of amplitude of respiration and rise in blood pressure on stimulation of the posterior parts of the orbital lobe.

Fall in blood pressure occurs immediately after the operation; however, this effect on blood pressure is not lasting because in the course of convalescence blood pressure returns to the pre-operative level and may even rise above it. However, Freeman and Watts⁹² noted that subjective symptoms related to high blood pressure are frequently reduced after lobotomy. Their clinical impression is that lobotomy may reduce the incidence of fatal hemorrhages in successfully lobotomized hypertensive patients. As to other circulatory phenomena, lobotomy was found to have been helpful to an obsessive patient in his convalescence from subacute bacterial endocarditis. Cold and clammy hands and feet become warm and dry, as if after a sympathectomy. Immediately after lobotomy there is considerable sweating. As for respiratory phenomena, lobotomy was found by Freeman and Watts⁹² to do away with anxiety that leads to hyperventilation, similarly to drugs that allay anxiety which likewise decrease the tendency to hyperventilation. Among gastrointestinal symptoms, a not infrequent finding throughout the period of convalescence, and sometimes outlasting it, is pathologic hunger. Vomiting occurs during the first twenty-four hours after the operation and appears to be particularly related to cutting of the medial basal fibers. Gastric acidity is increased early after lobotomy but becomes normal later. As to lasting changes, lobotomy appears to relieve pylorospasm immediately upon cutting the last frontal quadrant; this result usually remains sustained. Lobotomy appears also to give unspecific aid in certain forms of indigestion and dyspepsia. Lobotomy gives lasting relief to the spastic forms of colitis.

For several weeks after lobotomy there is an increased reactivity to drugs which stimulate the autonomic nervous system such as epinephrine and mecholyl, although the clinical psychotic syndromes may have been relieved (Rinkel and his coworkers¹⁴⁰). Later, however, there is an increased resistance toward drugs which upset the autonomic equilibrium (Reitman¹³⁸).

Electroencephalographic studies show the focal and generalized hypersynchrony expected after any brain lesion of this type. However, even markedly abnormal electroencephalograms tend to become normal two to four years after the operation. This restored normality applies only to the resting state of the electroencephalogram. A lasting dynamic change remains, namely a change in response to sleep, either natural or induced by barbiturates. Lennox and Coolidge¹⁰⁷ established the fact that the sleep spindles normally transmitted from the thalamus to the frontal lobes and from there to the remainder of the hemisphere (see also Brazier and Finesinger⁸⁸) are absent in the frontal lobes after lobotomy. My coworkers and I have confirmed this, finding also that such spindles are greatly reduced in the remainder of the brain as well.

Variants in Operation. In 1940 in discussing Mixter's⁸⁰ paper on lobotomy before the Boston Society of Psychiatry and Neurology, I suggested that if Kleist's¹⁰⁵ conclusions, based on his observations on mental changes following traumatic frontal lobe lesions, are correct, especially his conclusions regarding the social ego and other superego functions first considered in connection with the orbital areas by Leonore Welt,¹⁵³ cutting of the orbital fibers including the uncinate tract alone should be sufficient to produce the desired results. In 1945 Hofstatter and his coworkers,¹⁰⁴ acting on my suggestion that effective therapeutic results might be obtained by

limiting transection of the frontal fiber systems to the orbital areas alone, concluded that this modified operation equalled the results of the standard transection. The incidence of postoperative incontinence was thereby reduced from 50 per cent to 18 per cent. No epilepsy developed and dullness and slowing were less pronounced.

In 1948 Wilder Penfield and his associates¹²⁹ reported on the first results with gyrectomies, that is, excisions limited to cortical areas alone. These authors found that the desired effects were obtainable only with mesial and orbital excisions; lateral excisions alone failed to produce the desired effect of lobotomy.

In the same year Scoville^{143, 143a} initiated selective cortical undercutting in an effort to produce cortical lesions without interfering with the blood supply to adjacent areas of the brain. This method had the surgical advantage of direct vision, resulting in avoidance of injury of the veins and larger arterial branches. Scoville and his coworkers^{143a} found that undercutting Brodmann's areas 9 and 10 and the cingulate gyrus, as well as other combinations of undercutting to be taken up further below (see Chapter XII), were sufficient to produce the desired results.

More drastic surgery was undertaken by Peyton, Noran, and Miller¹³¹ who excised anterior portions of both frontal lobes (frontal lobectomy) with a similar rationale. They expected that such excision would leave no additional areas of cerebral tissue without blood supply to undergo subsequent necrosis. They also hoped that complete extirpation would assure lasting relief more regularly than lobotomy. They also advocated this measure because they could employ direct vision in the course of the operation; they could determine the exact quantity of tissue excised by weighing it and thus have no doubts as to the extent of the operation, and could study the tissue removed microscopically. Results in their series were less favorable than in comparable lobotomy series. They reported no full recoveries, one death occurred postoperatively, and 4 of the 11 patients in this series underwent epileptic seizures. However, it should be pointed out that all their patients had hopeless prognoses and lengthy duration of illness (one patient had been ill twenty-one years); and all had shown evidence of significant mental deterioration.

Mettler and his associates (pp. 75-76) reported in 1949 that bilateral removal of specific portions of the frontal lobes, namely Brodmann's areas 9, 10, and 46, produced a degree of improvement equal with that with lobotomies but did not create the same degree of unwanted side effects. In 9 patients in whom the three above-named areas were untouched by the operation no change in psychotic behavior took place.

Spiegel and his coworkers¹⁴⁷ produced by their method of *stereotaxic thalamotomy* lesions measuring 3 mm. in diameter, each covering about one-thirty-sixth of the volume of the dorsal medial nucleus of the thalamus. One or two such lesions placed in each dorsomedial nucleus of the thalamus had no or only a transitory effect in 3 patients. In those patients in whom five such lesions were produced in each dorsomedial nucleus, beneficial therapeutic results were obtained. As in frontal lobotomy, in the first stage following thalamotomy the patients exhibited somnolence, disorientation, chiefly for time, memory defect, inertia, and psychomotor retardation. Two patients had transitory weakness of one leg associated with a Babinski sign

and/or ankle clonus. In one patient with hypertension, blood pressure fell temporarily from an average of 190 systolic and 110 diastolic to 130 systolic and 95 diastolic. Soon after the operation appetite was frequently increased, leading to an increase in weight.

Surgery of the *temporal lobes* was initiated by Frederick A. Gibbs⁹⁸⁻¹⁰² discovery of specific spike foci located in one or both anterior temporal lobes in cases of psychomotor epilepsy, particularly in those in which rage-like outbursts of impulsive, brutal, aggressive, hostile behavior occurred. This discovery was of particular significance in view of the finding of Penfield and his coworkers^{21, 129a} that hallucinatory experiences predominated in the aura of patients with focal temporal epilepsy. Penfield and Rasmussen,¹³⁰ using their ingenious technique of stimulation experiments in the waking, cooperative patient, succeeded in producing hallucinatory experiences of not only acoustic but also optic, scenic, and dream-like memory elements by electric stimulation of the temporal lobes. Also various introspective illusions and experiences of somatopsychic dissociation were thus elicited and experienced with extraordinary vividness by the patients, who were wide-awake and merely under local anesthesia. Bailey and Gibbs⁸³ reported successful surgical treatment of psychomotor epilepsy by anterior temporal lobectomy in 1951. Obrador¹²⁷ had performed temporal lobotomy in schizophrenics with inconclusive results.

Thompson and Walker¹⁵¹ reported recently that extirpation of the *amygdaloid complex* in some disturbed schizophrenic individuals has resulted in greater placidity. Bard and Mountcastle⁸⁴ reported that wild animals, rats and monkeys, were tamed by amygdaloidectomy, while tame cats were made violent by the extirpation of this nucleus. Williams and Freeman¹⁵⁴ in a very interesting recent case report relief from a chronic hallucinosis by bilateral extirpation of the amygdaloid nucleus. In addition to the fact that the patient was relieved from his hallucinations, his general emotional attitude was benefited also: "whereas before the operation his attitude and posture were that of one carrying a heavy burden, he now strolled about the ward with the casual air of a bon-vivant. The many little characteristic shrugs and grimaces so frequently observed in everyday life among normal people [and which had been characteristically absent] reappeared. We could detect no elation or disorientation, apathy, or impairment of judgment. An air of reasonableness crept into his conversation which had not been present before. He denied hallucinations." Three weeks later "there had been some mild return of hallucinations but their insistent quality and general interference were greatly diminished. Six weeks after operation the patient returned to say that he was anxious to return to work and that the hallucinations recurred only when he was particularly fatigued and at bedtime. There seems to be little or no verbal substance to these auditory hallucinations, since they are now but indistinct sounds. He has developed an evenness of temperament and has shown enthusiasm for social activities, such as dates and larger group activities." Williams and Freeman¹⁵⁴ regarded this result as distinctly encouraging for further work along these lines. There is no doubt that the present trend of psychosurgery is in the direction of development of greater precision in physiologic interruption of specific fiber systems or extirpation of specific cellular aggregates.

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COMPLICATIONS AND FATALITIES DUE TO PHYSICAL TREATMENT METHODS

Prevention, Emergency Treatment and Lifesaving Measures

ELECTROSHOCK AND ELECTRIC STIMULATION TREATMENT

Circulatory and Respiratory Complications

Cardiovascular Stress Caused by Electroshock. The physiologic events which lead to complications and fatalities caused by circulatory disturbances—by far the most important set of complications encountered with electroshock treatment—were most succinctly and clearly described for animals by Urquhart¹²⁶ (p. 164) as early as 1927, more than ten years before the introduction of electroshock therapy: “The initial effect of the passage of the alternating current through the brain is intense stimulation of all the centers in its path. There is a generalized muscular contraction which, together with a pronounced vasoconstriction, results in a great accumulation of blood in the venous side of the circulation. The stimulation of the vagus center causes inhibition of the heart, an effect which, to a large extent, can be obviated by section of both vagi. That there is not total absence of inhibition of the heart with this procedure is evidence that there are extravagal inhibitory fibers, and when these are controlled with atropine there is no inhibition of the heart on the passage of the current.

“When the current is broken, this period of stimulation is followed by a period of profound inhibition or paralysis of all these centers. The animal becomes limp and unconscious; respiratory movements are absent; the eye reflex cannot be elicited; the blood pressure rises to almost twice the normal and then falls with the dilatation of the blood vessels even although the heart beats at an increased rate; there is no reflex response to sensory stimulation (stimulation of the central end of the cut sciatic nerve) and severe stimulation of the central end of the cut vagus has no reflex effect on the heart.

“That this paralysis may be only temporary is shown in our experiments by the fact that artificial respiration brought about recovery in all cases in which no gross damage to the brain or the spinal cord had been done by the heat generated at the point of application of the electrodes. That gradual recovery may follow the period of paralysis is clearly shown in charts 3 to 6. With the return of consciousness all the reflexes return, and in the majority of instances there is an interval of only a few seconds between the initial breath and their return.”

Urquhart's findings were independently confirmed by MacMahon.⁸³ By giving 1/100 to 1/150 grain of atropine to guinea pigs prior to low voltage electric shock from the mouth to a hindleg, he was able to prevent successfully the occurrence of cardiac failure and ventricular fibrillation. "When the heart was exposed and the same experiment repeated, we watched the heart continue beating while the current was passing through the body and we observed no change in rate; neither was there a pause nor irregularity after the current ceased. This was important for it showed that when the vagus was blocked with atropine, an electric shock, which in a control animal was sufficient to stop the heart, produce ventricular fibrillation, irregularity and acceleration, effected no appreciable response in either rate or rhythm."

Urquhart's classical study clearly differentiates two sets of factors in the cardiocirculatory disturbances created by electroshock: mechanical hemodynamic factors, and neurogenic factors.

Mechanical Peripheral Hemodynamic Factors, Resulting in an Accumulation of Blood on the Venous Side of the Circulation, and the Resulting Increase of the Inflow Load of the Heart. During the immediate post-convulsive phase this venous congestion greatly increases the inflow load on the heart, placing considerable stress on the myocardium. The reasons for this venous congestion are threefold. The first two mechanisms were referred to by Urquhart; they are (1) the pronounced arterial vasoconstriction, and (2) the convulsive muscular contractions, particularly the intermittent muscular contractions of the clonic phase. The third factor responsible for venous congestion is the Valsalva phenomenon, elicited by increase of intrathoracic pressure against a closed glottis. The importance of the Valsalva phenomenon in electroshock has been well described by Altschule and his coworkers.⁶ As the Valsalva phenomenon is relieved with the first breath at the termination of the seizure, the blood which has accumulated under considerable pressure in all venous channels rushes into the heart and greatly augments the heart's inflow load. At the same time, if vasoconstriction outlasts the seizure, the increased peripheral resistance which the heart must simultaneously overcome adds to the strain on the myocardium. The heart therefore may have to master a difficult situation consisting in a combination of increased inflow load with increased peripheral resistance, both of which occur during the minute or minutes immediately after the electrically induced seizure.

PROPHYLAXIS AGAINST EXCESSIVE MYOCARDIAL STRESS. Preventive protection against this stressful situation includes first of all the screening of those patients with myocardial weakness due to coronary heart disease or other factors. In this connection, however, it is important to keep in mind that no contraindication is an absolute one, with the exception of acute myocardial infarction (Moore,⁹⁰ Williams and Barrera¹³²), and the possible exception of severe mitral stenosis, since fairly effective measures are at hand by which this particular set of stress factors can be modified. The best of these measures is the use of the controlled modified convulsion, i.e., the convulsion rendered purely tonic by continued electric stimulation as described previously (Alexander²) (see Chapter IX). This type of seizure prevents excessive inflow load on the heart by the following means: first, by abolishing the intermittent contractions of the clonic phase, and second, by making possible—by means of gradual reduction of the stimu-

lating current—a more gradual relief of the Valsalva phenomenon, as manifested by a gradual lowering of intrathoracic pressure during the convulsion itself (see Fig. 89, Chapter XIV). This prevents the sudden influx of venous blood after the clonic phase, such as occurs during uncontrolled tonic-clonic seizures. The controlled tonic seizure also prevents the increase in peripheral resistance otherwise occurring during the clonic phase and lasting through the immediate postconvulsive period; when the clonic phase is eliminated, the peripheral vasoconstriction and pilomotor erection which are usually associated with and generally outlast this phase are eliminated as well. This is probably due to inhibition of the central sympathetic vasoconstrictor and pilomotor centers, which are inhibited together with the clonic phenomena. It thus appears that the mechanical and peripheral types of cardiovascular stress can be prevented by the use of the purely tonic seizure. I have employed it in a number of patients with coronary heart disease without incurring a single mishap.

Neurogenic Central Factors Causing Disturbance of Heart Action, and Prophylactic Measures. The other set of factors constituting stress on the cardiovascular system is neurogenic and is due to the excessive excitation and subsequent inhibition of the vagus; this was clearly proved by Urquhart¹²⁶ and MacMahon,⁸³ as quoted above. This neurogenic factor plays a role particularly in individuals with slow heart rate or with prolonged P-R interval, as well as in the general group of vagotonic individuals. Administration of 1/50 grain of atropine fifteen minutes before treatment is the best prophylactic measure.

In patients with marked tachycardia which interferes with coronary circulation and causes a certain amount of myocardial ischemia—especially in patients with sinus tachycardia, and particularly in those with auricular fibrillation—pretreatment with quinidine is essential: 12 grains (0.8 gm.) daily for two or more days prior to treatment and throughout the intensive treatment phase is indicated (Hayman,⁶⁰ O'Flanagan and Taylor⁹⁶). As the patient improves, and electroshock treatments are spaced more widely, quinidine given for one day prior to and on the day of treatment generally suffices. When it is merely a problem of uncomplicated tachycardia, 6 grains of quinidine taken by mouth twenty minutes before treatment is usually sufficient.

Cardiovascular Accidents due to Electroshock Therapy. The cardiovascular accidents which may occur during shock treatment and may endanger the life of the patient fall into the four following groups:

(1) *Myocardial Failure and/or Infarction (Acute Heart Failure).* This may be due to myocardial degeneration without coronary heart disease, as in the case of a 62 year old man described by Napier.⁹⁴ In other cases, this may be related to severe coronary heart disease without infarction, as in the case described by Cash and Hoekstra²² and in a case described by Jetter.⁶⁶ Death in these two instances occurred within three and one-half minutes and two hours respectively. In two similar cases (Jetter and Eyman and Morris³⁴), old infarcts were found as well. Death occurred in these cases immediately and twelve minutes after treatment respectively. In two other cases described by Eyman and Morris³⁴ and by Ebaugh, Barnacle, and Neubuerger,³⁰ fresh coronary occlusion took place within three-quarters of an hour to one and a half hours after shock treatment. In

a case described by Martin,⁸⁶ myocardial infarction occurred shortly after shock treatment, causing death eighteen days later.

(2) *Vascular Collapse*. This may take place under the influence of excessive vagal stimulation combined with peripheral vasodilatation, not only following convulsive electroshock therapy, but also after nonconvulsive or myoclonic electric stimulation treatments. If promptly treated, peripheral vascular collapse need not be fatal. Prompt injection of 0.3 to 1 mg. of epinephrine intravenously, or, if necessary, into the heart or a large intrathoracic vessel, will promptly resuscitate the patient.

A syndrome of peripheral vascular collapse with profuse perspiration, following convulsive electroshock treatment, in one of my patients was demonstrated to have been caused by functional hyperinsulinism. The hypoglycemic attack was precipitated by anxious anticipation combined with inadequate food intake for more than 30 hours prior to treatment and by the violent muscular exercise incidental to the treatment itself. Adequate psychotherapeutic preparation and nursing care in the hospital prevented recurrences in this case, and the patient was able to complete his treatment series uneventfully until he attained complete recovery from his psychosis.

(3) *Pulmonary Edema*. The causes of pulmonary edema are not completely understood, but when this condition occurs in connection with electroshock treatment, it is probably attributable to left ventricular failure. Other causes may be mitral stenosis or neurogenic vagal disturbances, as well as osmolar disturbances due to endocrine factors. A number of other causes, including hypothalamic lesions, have been enumerated and studied by Gamble and Patton.⁴⁵ The literature includes 5 cases in which death ensued from pulmonary edema after electroshock within twenty minutes to two days after treatment.^{37, 54, 66, 120, 131} The management of this life-threatening emergency will be described in detail further below.

(4) *Disturbances of Heart Rhythm*. These are in large measure due to the vagal disturbance described previously. They may range from simple sinus tachycardia to auricular or ventricular fibrillation. A fatal case of this type was described by Ebaugh et al.³⁰ Arrest of the heart lasting for as long as 46 seconds after an electrically induced convulsion is not infrequently observed without subsequent ill effect, especially if respiratory activity is not impaired during that critical phase. This emphasizes the great importance of immediate reestablishment of respiration following the seizure, because, if anoxia is added to a severe disturbance or cessation of cardiac rhythm, the heart muscle may be irreversibly damaged. Premedication by atropine will frequently prevent the arrest of the heart. Premedication by quinidine may prevent sinus tachycardia and auricular fibrillation.

Respiratory Accidents. The most important of these is respiratory failure. Three fatal cases of this type are described in the literature (Ebaugh et al.,³⁰ Smith, Hastings, and Hughes¹¹⁹). In two of these cases, curare had been employed. Generally, respiration can be promptly reestablished by nonconvulsive stimulation or by artificial respiration, a few lifting movements of the thorax usually sufficing. After convulsive treatment with an alternating current machine, unsuitable for nonconvulsive stimulation, artificial respiration should be applied immediately after the convulsion

unless adequate deep respirations spontaneously reestablish themselves promptly upon the termination of the seizure. The same should, of course, be applied after treatment with unidirectional current, if shift to nonconvulsive stimulation fails to reestablish adequate respirations immediately.

Laryngeal spasm is far less frequent than is generally assumed. In most cases, what clinicians too readily assume to be laryngeal spasm is actually occlusion of the glottis caused by the falling back of a relaxed large tongue. This can be prevented by prompt insertion of an airway at the termination of the seizure (see Chapter XIV).

Cases of pneumonia as well as of pulmonary infarction, usually due to aspiration of vomitus, mucus or saliva during the postconvulsive phase, have been described.^{11, 30, 32, 86} Patients who salivate excessively owing to the parasympathetic stimulus of electroshock should be given 1/100 to 1/50 grain of atropine before treatment.

Other Circulatory Complications, Including Cerebral Complications. An unusual case of extensive hemorrhages into the thyroid leading to death within six hours has been described by Napier.⁹⁴ It seems likely that these hemorrhages occurred in some relation to increased venous pressure, secondary to the Valsalva phenomenon. This may also have been the case with some cerebral complications, as in such nonfatal cases which developed cerebral vascular lesions. Illustrative cases of nonfatal hemiplegia were described by Kaldeck⁶⁷ and Olsen.⁹⁸ Fatal cases with pericapillary hemorrhages, mostly in older persons, have been reported by Riese¹⁰⁹ and Gralnick.⁵⁵ In addition, Martin⁸⁶ has reported pericapillary intracerebral hemorrhages in the case of a young malnourished patient suffering from chronic pyloric stenosis. In two other cases, death ensued during status epilepticus, which was in one instance further complicated by hyperpyrexia. Death occurred in these two cases within three days and one week respectively after electroshock therapy; unfortunately, in neither case was an autopsy performed (Gralnick⁵⁴ and Ebaugh et al.³⁰). Meyer and Teare⁸⁸ described an important case in which death resulted from a fat embolism after possible vertebral compression fracture twelve hours after the patient's first and final electroshock treatment.

Wassersug and McLaughlin¹²⁸ point out that tuberculosis is not a contraindication to electroshock therapy. In the two cases reported in the literature in which fatalities following electroshock were attributed to tuberculosis (Martin⁸⁶ and Napier⁹⁴), death occurred six months after the last electroshock treatment. Obviously electroshock bore no direct relation to the cause of death in these cases.

Emergency Equipment. Emergency equipment available for all patients undergoing electroshock therapy should include: oxygen, airway, tracheal tube, suction, four rubber tourniquets, and the following drugs: epinephrine, in ampules containing 1 mg. in 1 cc. of water; coramine in 2 cc. ampules; aminophylline in ampules containing 250 mg. in 10 cc. of water; quinidine for intramuscular injection in 0.6 gm. doses; neo-synephrine for injection in a 1 per cent solution; paredrine in ampules containing 10 mg. in 1 cc. of water; atropine in hypodermic tablets containing 1/100 grain each; and morphine in the form of 1/4 grain hypodermic tablets.

The Management of Cardiovascular Complications and Pulmonary Edema. If any difficulty in circulation is encountered, oxygen by in-

halation should be given immediately as the first resuscitation measure. In addition, the following treatments should be resorted to, depending on prompt diagnosis of the difficulty.

If the heart beat is of poor quality, indicative of impending circulatory collapse, 0.3 cc. of a 1:1000 solution of epinephrine should be administered intravenously, to be repeated if and when necessary. In actual shock due to peripheral circulatory collapse, the standard treatment should be instituted immediately, including elevation of the feet, administration of 0.5 to 1 cc. of a 1:1000 solution of epinephrine intravenously, and/or 1 to 2 cc. of coramine, given intramuscularly or intravenously depending upon the urgency of the situation, or 0.3 to 0.5 cc. of neo-synephrine or 0.5 cc. of paredrine to be repeated every fifteen minutes.

If consistent heart pain, indicative of constriction of the coronary vessels, manifests itself, or in case of coronary occlusion or cerebral vascular accidents, intravenous injection of aminophylline is in order; 250 mg. in 10 cc. of water should be administered at the rate of 2 cc. per minute.

If rapid pulse or arrhythmia, auricular or ventricular fibrillation develops, 0.6 gm. of quinidine should be administered intramuscularly.

If pulmonary edema should manifest itself by dyspnea and frothy and bloody sputum, the patient should immediately be elevated to a sitting position, feet hanging downward over the side of the bed, with the simultaneous administration of oxygen. It is useful to add ethyl alcohol vapors to the oxygen (Gootnick et al.⁵⁰). The addition of ethyl alcohol inhalation as an antifoaming agent aids in the elimination of the froth in the broncho-alveolar spaces with resulting improvement in conditions for gaseous exchange. Gootnick and his coworkers described a simple and effective technique for combining such inhalation with pressure oxygen therapy by means of ordinary equipment. These emergency measures alone may control the situation in milder cases. If the disturbance is persistent, tourniquets should be applied to three extremities at a time, to be rotated clockwise every fifteen minutes. One-fiftieth grain of atropine and $\frac{1}{4}$ grain of morphine should be given. If the blood pressure declines, neo-synephrine or paredrine must be administered. Prompt management of this type was successful in bringing about recuperation within eight hours of a patient suffering from pulmonary edema following electroshock treatment. I am greatly indebted to Dr. George S. Kurland for his valuable contribution to the successful management of this case.

The patient was a 25 year old male suffering from a schizo-affective psychosis with marked anxiety. There was no previous history of cardiac disease. Labile hypertension had been noted by his family physician; growing pains had occurred during childhood. He had served in the armed forces, and no cardiac disease had been noted. Four nonconvulsive treatments had been administered previously without unusual incident. The fifth treatment was a combined convulsive-nonconvulsive treatment of three minutes' duration administered through temporo-parietal leads. The treatment itself was well tolerated; the patient then rested for twenty minutes, awaiting psychotherapy, when it was noted that he was dyspneic, ashen pale, and perspiring. There was a rapid progression of symptoms with the development of cyanosis, increased dyspnea, and grossly audible rales. The blood pressure at that time was 150/80. As soon as the diagnosis of pulmonary edema was made, i.e., within a few minutes after the onset of the disturbance, the patient was placed in an upright sitting

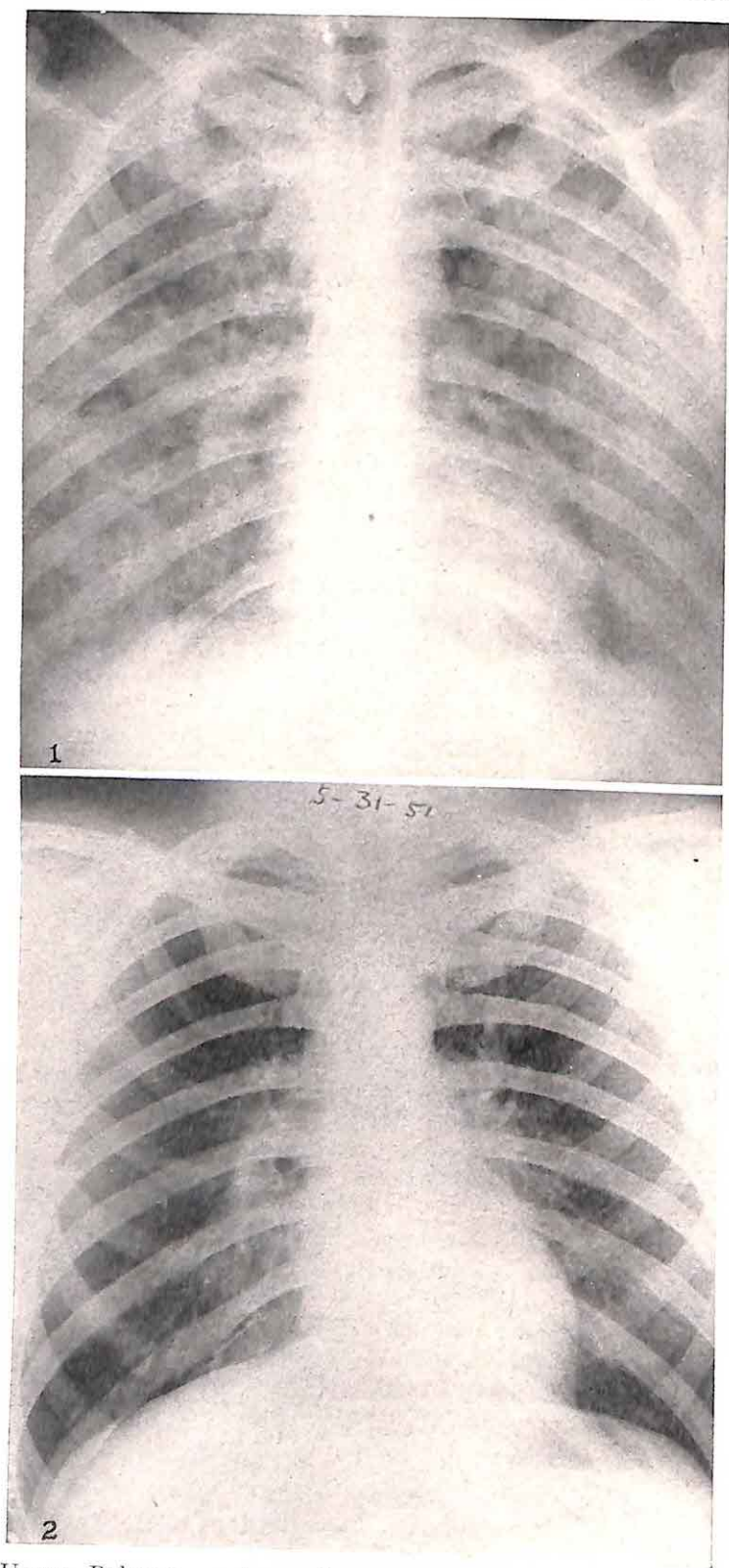


Fig. 45. Upper, Pulmonary edema after electroshock treatment. X-ray taken four hours after onset. Lower, X-ray taken three days later, showing recovery from the disturbance.

position, oxygen was administered, atropine 1/50 grain and morphine 1/4 grain were given subcutaneously and tourniquets were applied to three extremities at a time. When the medical consultant arrived one hour later, the blood pressure had declined to 120/90, the patient was cyanotic, perspiring profusely, dyspneic, orthopneic, and in obvious acute distress. He was coughing up frothy pink sputum and vomiting chocolate brown material. The radial pulse was weak and thready. The neck veins were not distended, nor was the heart enlarged. The rhythm was grossly irregular with an apical rate of 180; the heart sounds were obscured. Both lungs continued to show moist bubbling rales. Neither liver nor spleen could be felt. An electrocardiogram showed auricular fibrillation at a rapid rate.

Treatment was continued with morphine, oxygen, and tourniquets. There was some clearing of the pulmonary edema, but the blood pressure fell slowly. A second electrocardiogram showed sinus tachycardia and ST segment changes. The blood pressure became imperceptible despite the administration of 10 mg. of paredrine intramuscularly and orally. The patient was transferred by ambulance to a general hospital while the administration of oxygen was continued en route. At that time, the pulse was 160-180; blood pressure was unobtainable; an x-ray of the lungs taken with a portable machine showed pulmonary edema (Fig. 45, upper); the general condition otherwise remained unchanged. Oxygen therapy was continued; the tourniquets were gradually removed. Lanatoside C 0.8 mg. was administered intravenously. The pulse rate slowed to 125, and over the course of the subsequent five hours, the blood pressure rose gradually, the limbs became warm and dry, and the lungs cleared of rales. Throughout the period of emergency, the patient had remained mentally clear and conscious, although later he manifested amnesia for the period extending from the electric treatment to the time three hours later when he was placed in the ambulance.

The following day, the patient complained only of sore throat, headache, and cough. Both lungs were filled with expiratory wheezes. With symptomatic therapy and rectal aminophylline, the symptoms gradually cleared. More extensive examination of the heart revealed no abnormalities; the x-ray showed normal size and configuration; the electrocardiogram was normal. An x-ray of the lungs three days after the incident was normal (Fig. 45 lower). The blood pressure was labile and varied from 130 to 160 systolic. The basal metabolic rate was minus 6. An upper gastrointestinal x-ray revealed no abnormality. The patient was discharged as recovered on the eighth hospital day. He also achieved a significant remission from his psychosis lasting for six months after this treatment, when anxiety and delusional preoccupations recurred.

Mortality Rate. It is interesting that in spite of these obvious dangers of electroshock therapy, the fatality rate is remarkably low. Deshaies and Pellier²⁷ have published a very excellent survey of the literature as well as of their own experiences with electroshock. These authors report a mortality rate of 0.08 per cent of patients, and 0.008 per cent per total number of treatments, based on 16,685 patients culled from the literature. The subgroups which composed this figure are in substantial agreement (see Table 3).

Musculoskeletal Complications

The most important of the other complications occurring during electroshock treatment are those relating to the musculoskeletal system. As an early paper by Friedman, Brett, and Vogt⁴² pointed out, vertebral compressions occur almost exclusively when the vertebral column is in a

flexed position, a situation they remedied during metrazol therapy by placing the patient's head and upper trunk at the foot of a hospital bed and raising the lever that normally permits flexion of the knees. This position provided adequate hyperextension of practically the entire spinal column and, together with manual pressure at the shoulders during the convulsive reaction, achieved a maximal amount of intervertebral spacing. While it is obvious that proper posturing, absence of excessive restraints during treatments, as well as a slow raising of the stimulating current (glissando technique—Tietz et al.¹²⁴) are capable of reducing the incidence of fractures, it is certain that in clinical practice, they cannot be entirely avoided. The general incidence of such complications has been well summarized by Deshaies and Pellier, who found from reports in the literature that osteoarticular accidents occurred in approximately 1.4 per

Table 3. Mortality Rates in Electroshock Therapy

SOURCE	RATE %	NUMBER OF PATIENTS
Müller ⁹¹	0.07	1363
Kolb and Vogel ⁷⁴	0.06	7200
Jetter ⁶⁶	0.12	2500
Moore ⁹⁰	0.1	2181
Nairac ⁹³	0.1	2000
Evans ³³	0.13	750
Laboucarić ⁷⁸	0.2	450
Deshaies and Pellier ²⁷	0.18	565
Kalinowsky ⁶⁸	0	1500
Delmas-Marsalet ²⁵	0	5000

cent of all treated cases. Jaw dislocations were common, occurring in 6 per cent of the reported cases; shoulder dislocations occurred in an average of 0.5 per cent; jaw fractures in 0.4 per cent; fractures of the humerus in 0.18 per cent; fractures of the femur in 0.2 per cent. The rate of occurrence of vertebral fractures derived from these authors' data is 1 per cent of patients treated, or 0.1 per cent of the total number of shock treatments administered. We must, of course, differentiate between symptomatic and asymptomatic vertebral fractures, which may well account for the divergencies in percentages reported by different authors.

Protective Measures after Fractures, Especially of the Vertebral Bodies.

Once such a fracture has occurred, treatment can generally be continued, provided the fractured part is given protection in the form of mechanical support, as well as with the additional safeguard of taking care to soften the procedure. The introduction of the first of these general measures for protection—curare—was made by Bennett,¹⁶ who administered intravenously an aqueous solution of intocostin containing 10 mg. of active curare principle per cubic centimeter, over a period of one to two minutes. He recommended administration of 1 cc. per 15 to 20 pounds of body weight. In most cases, however, 1 cc. per 40 pounds of body weight is sufficient. Ephedrine, 25–50 mg. in 1 cc. of water, should be added to the intravenous injection. Prostigmine methylsulfate, 0.5 to 1.0 mg., should be given intramuscularly immediately upon completion of the seizure. If spontaneous respiration is not resumed immediately, artificial respiration must be given, preferably with administration of oxygen under positive pressure. Airway must be inserted, and a tracheal tube ready when needed.

The risk of curare is augmented by the fact that it enhances the vagal stimulating effect of electroshock (Altschule and Tillotson⁷); furthermore, subclinical states of myasthenia gravis may enhance and prolong its respiratory paralyzing effect (Beard and Harris,¹³ Brody¹⁹).

Another similar drug is decamethonium bromide (C 10) (Holt, Rinkel, Nicholson and Solomon⁶³), but it is potentially more hazardous because of its more profound paralyzing action upon respiration. Myanesin presents even greater difficulties, inasmuch as the margin of safety between an effective paralyzing dose and a lethal dose is very small; it also raises the convulsive threshold, requiring an excess of current in order to produce a therapeutic seizure (Holt, Rinkel, Greenblatt and Anderson⁶²).

Pentothal sodium given intravenously in the same manner as for non-convulsive stimulation (see Chapter XIV), i.e., about 6 to 7½ grains (0.5 gm.) administered fairly rapidly until the patient drops off to sleep, will soften the convulsion almost as well as curare and its derivatives, and has the advantage of eliminating the unpleasant progression of paralysis which the patient experiences consciously when curare or its derivatives are used. It also constitutes a lesser hazard to the resumption of adequate respiratory activity after the shock. Mastery of the technique of purely tonic convulsions will greatly reduce the need for either curare or pentothal sodium, although the latter will remain useful in eliminating pain when the patient makes brusque movements during the cloudy postconvulsive state. Purely tonic convulsions constitute a further advantage if, after a vertebral fracture has occurred, they are administered in a removable body cast, thus eliminating any danger of dislocation.

Other Clinical Complications

Among the rare complications it has been observed that arthritis may become aggravated after electroshock (Evans³¹). This is not surprising in view of the reciprocal antagonistic effect of electroshock therapy and ACTH and cortisone respectively, as described by Altschule et al.,⁵ Rome and Braceland,¹¹² and others.

Gastric ulcer may also become aggravated by convulsive electroshock (Evans³¹), presumably in view of the vagal stimulation incidental to electroshock. It is important that patients fast for at least four hours prior to electric treatment because of the danger that the patient may vomit before his cough reflex is fully reestablished (Baur and Perrin¹²).

Subconjunctival hemorrhages are occasionally observed with electroshock; their occurrence may denote a lowering of capillary resistance incidental to nutritional deficiency. Ample supplies of vitamin C (100 mg. three times a day) and rutin (60 mg. three times a day) will generally prevent their recurrence.

Electroshock, especially if tonic convulsions are used, but even if full tonic-clonic convulsions are given, can be administered to pregnant women without ill effects on the fetus (Polatin and Hoch¹⁶³). Pregnancy does, however, contraindicate insulin coma treatment (Gralnick⁵⁶).

Organic diseases of the nervous system including multiple sclerosis do not constitute contraindications to electroshock therapy when its use is indicated for the treatment of coincidental depression (Alexander,¹ Savitsky and Karliner¹¹⁴). In multiple sclerosis, remissions have sometimes been initiated simultaneously with the relief of a depression by electro-

shock treatment. In cases uncomplicated by depression, however, electroshock has been ineffective in influencing the course of the disease.

INSULIN COMA TREATMENT

Irreversible Coma

The most important complication of insulin coma treatment is irreversible coma. Patients who lapse into this state either may die during the subsequent hour or few hours in a deep medullary phase of the coma, or they may survive for as long as eight weeks in a comatose, semicomatose, stuporous, deliriod, or decerebrate state. In the course of this terminal state, patients may exhibit the signs and symptoms of severe irreversible brain damage, such as impulsive motor overactivity, tetraplegia, astasia-abasia, hyperthermia, and other syndromes of decerebration. Survival, and remarkable though varying degrees of recovery of brain functions, however, may still ensue after as long as six days to two weeks of coma. Goldman⁴⁸ has reported an illustrative case, that of a woman who survived two weeks of "irreversible" coma, followed by three months of a purely vegetative existence during which time she was spoon fed. Metrazol treatment, employed as a desperate last measure, improved her progress to a sitting and walking state. This patient eventually regained her power of speech, but had complete amnesia for the past, and no return of orientation was manifested. Numerous cases are reported in the literature of comas prolonged as much as six days from which the patients recovered with only transitory ill effects.^{23, 40, 47, 51, 105}

These accidents make it imperative that the physician bend every effort to avoid this complication, and that once it has occurred, he make every effort to resuscitate the patient as promptly as possible in order to prevent the accretion of any avoidable brain damage. In a case of my own experience, a patient was successfully revived from "irreversible" coma within twenty hours; and by taking adequate precautions for oxygenation, glucose supply, supply of cocarboxylase and other metabolic catalysts in the form of vitamins, brain damage was completely avoided.

Causes of Irreversible Coma. Among the 58 well documented fatalities reported in the literature*, 36 are cases of irreversible coma. Analysis of these data emphasizes two outstanding causes of irreversible coma: faulty dosage and too long duration of coma prior to attempted interruption.

Faulty dosage may involve the following mechanisms: (1) Too large an initial dose, as in a case described by Malamud⁸⁴ in which 50 units were used in the first treatment. (2) A dose which is too rapidly raised from treatment to treatment as illustrated in a case reported by Scheffen¹¹⁶ in which six treatments were given employing the following doses: 20, 80, 160, 250, 300, 450 units, and, after an interval of twenty days, resumption of treatment with a dosage of 600 units which proved fatal. (3) A large dose given repeatedly, without consideration of the fact that in the course of treatment the patient may display sensitivity to that dose, may also result in fatal irreversible coma. In one reported case of a patient¹¹⁶ for whom termination had been difficult the previous day after a dose of 450 units, a decrease in dosage was obviously in order; however, the patient received the same dose on the subsequent day, resulting in fatal irreversible

* See refs. 8, 9, 15, 17, 21, 28, 38, 39, 43, 44, 53, 59, 64, 70, 73, 76, 79, 80, 82, 84, 87, 92, 99, 104, 106, 107, 115, 116, 117, 118, 121, 127, 135.

coma. In another case discussed by the same author, a strangely erratic series of dosages set the stage for sensitivity and irreversible coma. Treatments were given employing the following doses on ten subsequent days: 50, 100, 200, 400, 800, 1600, 1600, 2000 and 500 units.

Fatal irreversible coma has also resulted in a number of cases when interruption of coma was not attempted in the face of such danger signs as a slowing of pulse during the deepest medullary phase of coma, or the appearance of a convulsive seizure, which must always be considered an indication for immediate termination of the coma by the intravenous route. Schefflen¹¹⁶ has noted a case in which routine termination was not instituted within seventy minutes of coma—which exceeds by ten minutes the maximum safe duration of coma—and hence proved fatal. In another case reported by this author, the third coma was interrupted by gavage after one hour. The third coma should never be permitted to exceed three-quarters of an hour when interrupted by the intravenous route, or one-half hour when gavage is employed, since the latter method requires an average time of ten to fifteen minutes to take effect. When dyspnea developed in this same case, after 125 gm. of glucose had been given by vein, and torsion spasms set in, the therapist failed to recognize these spasms as the symptoms of tetany, and therefore did not treat them as such. Intravenous injection of 30 cc. of 10 per cent calcium chloride is indicated in such cases.

Prevention of Irreversible Coma. Irreversible coma is best prevented by such measures as the gradual increment of the dose, by no more than 20 units over the preceding day's dose, and by initiating the treatment series with a dose as low as 10 units, as well as by prompt lowering of the dose when signs of excessive susceptibility develop. As soon as signs of increased sensitivity and reactivity to insulin become evident, such as early precipitous depth of coma or delay and difficulty in arousal from coma, the subsequent day's dose should be lowered by 15 or 20 units, or, better, by about 15 per cent of the dose of insulin given. The most important single factor in preventing irreversible coma is the prompt termination of all comas after no longer than fifteen minutes of the medullary—maximally deep—phase (see p. 131).

Treatment of Irreversible Coma. Resuscitation measures in the event of irreversible coma consist first of all in prompt diagnosis of the condition. Time must not be futilely spent in the hope that the patient may still arouse in response to the obviously inadequate routine dose of glucose. If the coma has not been terminated within five minutes after the original attempt at intravenous interruption, or within twenty minutes after gastric gavage was employed, an additional dose of 25 gm. of glucose should be promptly administered, this time always by vein. If the patient fails to regain consciousness within five minutes after this additional dose, the diagnosis of irreversible coma must be made, and the *full regimen for relief of this condition* as outlined below must swing into action:

1. The most vital aspect of this regimen is the continued supply of adequate amounts of glucose so as maximally to facilitate uptake on the part of the brain. As soon as possible, continuous intravenous drip infusion of the brain. As soon as possible, continuous intravenous drip infusion should be initiated, administering 10 per cent glucose in saline. This should be supplemented by repeated additional injections of 25 per cent glucose through the infusion tube, sufficient to bring the hourly administered dose of dextrose to 50 gm. Addition of concentrated dextrose is par-

ticularly helpful whenever the need seems indicated by a deepening of hypoglycemic symptoms, such as the recurrence of profuse perspiration after it had once abated following the previous administration of hypertonic dextrose. The therapist should not be deterred from continued administration of dextrose during this period by the fact that after a time during irreversible coma, the blood sugar may return to its normal level. The vital fact that the ability of the brain to utilize glucose is sharply reduced both during coma and subsequent to coma must always be kept in mind (Loman^{81a}). For this reason it is essential that an overabundant supply of glucose be offered continually during this crucial phase. The need for this is clinically quite striking in such cases, although the mechanisms involved are undoubtedly more complex than is generally appreciated at present. As a general rule, it may be stated that the total amount of glucose in grams which must be supplied within the first twenty-four hours of irreversible coma is approximately three to four times that of the dose of insulin in units which had been given. Continued administration of glucose is also needed during this period because intramuscular pools of such large amounts of insulin as are used in insulin coma treatments appear to remain active for up to eighteen hours or more, depending on the amount of the dose (depot-effect), and because as a result of the rise of the blood sugar level during resuscitation by glucose, the patient's own pancreas, or rather his entire parasympatho-insulin system, becomes stimulated to activity, i.e., to additional production of insulin which again abrogates the brain's priority over the circulating glucose. For this reason it is necessary to assure abundant supplies of glucose to the brain throughout the period of resuscitation by continued administration. While, as a general rule, 50 gm. per hour should be given by vein through the first twenty-four hours of coma, this dose can be modified later in accordance with the ability of the patient to take other forms of nourishment. With such prompt and adequate treatment, however, most cases of prolonged coma will be resuscitated successfully within the first twenty-four hours.

2. Thiamine chloride and other vitamins, particularly convenient in the form of ampules of Lyo-B-C (Sharp and Dohme), should be added to the infusion fluid, in order to supply enzyme systems (particularly cocarboxylase which is diphosphothiamine) necessary for the oxidation and utilization of sugar by the central nervous system.

3. The patient should be placed in an oxygen tent to facilitate oxygen supply to the central nervous system, and to ease respiration, thus preventing exhausting hyperkapnia and its complication, hyperventilation tetany.

4. As soon as the patient has been resuscitated from the deepest (medullary) level of coma (during resuscitation the patient passes through the successive phases of coma in reverse order), he should be kept warm with the aid of heating pads or warm water bottles in order to guard against vascular collapse.

5. If signs of tetany, usually due to hyperventilation, develop, 30 cc. of 10 per cent calcium chloride should be added to the infusion fluid or injected independently.

6. Respiratory or circulatory stimulants may be added if and when the condition of the patient requires them.

Additional measures such as blood transfusions have been recommended

(Wortis and Lambert¹³⁴), using about 500 cc. of citrated blood. The administration of 3 to 10 cc. of adrenal cortical extract in order to counteract what appears to be a transient deficiency of this hormone has been suggested by Freudenberg⁴¹ and Gottesfeld.⁵¹ Goldman⁴⁸ recommended repeated intravenous administration in doses of 3, 5, or 10 cc. Clower and Niswander²⁴ gave 200 mg. of cortisone in four divided doses intramuscularly, or 100 mg. once daily by gavage tube. These authors, as well as Hoffman and Wunsch,⁶¹ also recommended the application of repeated bursts of non-convulsive electric current, lasting 10 seconds each and separated by intervals of 10 to 20 seconds. If six such current applications fail to bring about some degree of improvement in the patient's condition, they feel that further stimulation will prove unsuccessful in lightening the depth of the coma or awakening the patient, and the therapist must then revert to other measures for resuscitation. Clower and Niswander also suggest elective tracheotomy in cases of severe respiratory embarrassment, pointing out that when tracheotomy is considered, the decision should be made as promptly as possible, so that it may be performed electively rather than as an emergency procedure later when the patient's general condition may have worsened. Sullivan¹²³ has recommended spinal drainage to be carried out routinely every eight hours during the course of prolonged coma, in addition to the routine administration of 0.3 cc. of a 1:1000 solution of epinephrine hydrochloride hypodermically immediately upon the appearance of a convulsion. I am, however, firmly convinced that this technique is injurious rather than helpful, a view in which Kalinowsky and Hoch⁶⁹ (p. 53) concur.

Other Complications

Cardiovascular and Respiratory Complications. Second in importance among the causes of death following insulin coma therapy are cardiovascular disturbances, including pulmonary edema. Cardiovascular accidents accounted for 10 deaths in the literature, pulmonary edema for 5. In this group of patients, excessive insulin dosages may have played a contributory role. MacKeith and Meyer⁸² have reported a case which may also be regarded in part as due to difficulty of termination of the coma. This patient obviously suffered his cardiovascular collapse during a secondary coma which followed shortly after a difficult but successful termination. There are, however, reports of cases of pulmonary edema (Young et al.¹³⁵) and circulatory failure and collapse not related to irreversible coma (Lawrence et al.⁷⁹ Barnett,⁹ Scheffen et al.¹¹⁶).

The best treatment of cardiovascular disturbances, is, of course, preventive treatment, which involves careful examination of the patient prior to the institution of insulin coma therapy, careful avoidance of excessive dosage, and prompt interruption by intravenous administration of glucose whenever severe untoward symptoms occur during the course of the therapy. This last factor alone may relieve the disturbance.

Obstructions of the throat should be treated by proper positioning of the patient's head and by suction. Any disturbance of respiration or circulation other than purely mechanical ones requires interruption of the coma as the first therapeutic step. Other measures may be instituted as needed, similar to those employed following electroshock. Coramine may be used as a

general analeptic and respiratory stimulant, epinephrine in the treatment of vascular collapse. Paredrine or neo-synephrine together with upright posture, oxygen with or without alcohol vapors, application of tourniquets, and the administration of atropine and morphine are indicated for the relief of pulmonary edema. The administration of 10 gm. of potassium chloride prior to treatment has recently been recommended (Kraft, Salzberg, and Rosenkrantz⁷⁵) as a means of preventing certain side effects of insulin upon the heart muscle, such as are demonstrated by a depressed T wave and sagging of the ST segment.

Complications of Interruption by Stomach Tube and Prophylactic Measures. A number of cases have been reported in the literature in which death ensued due to pneumonia, which in turn was brought about by aspiration of the gavage material or of vomited nasal tube material.^{44, 59, 99, 115, 117, 129} When the nasal tube method of termination is employed it is important, in order to check the position of the tube, to withdraw stomach fluid by suction and identify it as such by testing its acidity with litmus paper before the glucose solution is administered. This precaution should be employed routinely, since, during coma, the cough reflex is abolished. The gavage material should be warmed to body temperature before it is administered both in order to avoid irritation of the membranes of the stomach and to facilitate more rapid absorption. When the tube is withdrawn, it should be tightly closed off in order to avoid the possibility of aspiration at this time. Every precaution must be taken to avoid such a complication, and its possible sequelae, pneumonia and pulmonary gangrene.

Secondary Comas: Their Prevention and Management. A fourth important group of fatalities are those occurring during secondary comas. These are particularly treacherous when they occur at night because they tend to go unrecognized until morning and thus easily deepen into irreversible comas (Ferraro,³⁸ Murphy⁹²). The occurrence of such comas may be greatly decreased, of course, by adequate interruption of the initial coma. If 1 gm. of glucose is administered for every unit of insulin that was employed, secondary comas will occur rarely if at all. If large amounts of insulin have been given, however, it is frequently not feasible to supply an equal amount of glucose. The therapist must occasionally rely on the patient's consumption of an adequate midday meal in order to attain a proper balance. Secondary comas sometimes occur when such patient's appetites wane, and they neglect to eat the precise amount of food required, while at the same time this lack may escape the attention of the nurse who might otherwise supply additional glucose or sugar. More than the usual amount of exercise which had not been taken into account by the attendants may also create a negative balance. One of my patients lapsed into such a secondary coma on returning from an unscheduled walk with a newly assigned occupational therapist.

If the staff is on the alert for such secondary comas, no harm will ensue. If a state of secondary hypoglycemia is discovered on the basis of undue drowsiness or perspiration before it has deepened into coma, administration of 100 gm. of glucose in orange juice by mouth will generally prevent coma. If the patient has actually lapsed into secondary coma, the intravenous route of resuscitation by dextrose should be used exclusively, since the precise onset of the coma can frequently not be ascertained and unnecessary

additional time should therefore not be lost. The response to immediate intravenous administration of 25 gm. of dextrose is usually prompt. If not, the full regimen for resuscitation from irreversible coma is indicated.

However, if such secondary comas remain undiscovered, as in the case of a patient taking a nap by the fireside where the telltale beads of perspiration may not immediately arouse suspicion, the patient is in danger of lapsing into irreversible coma. For this reason, secondary comas are particularly dangerous when they occur at night and especially during warm summer nights when perspiration may be so common as to fail to impress itself on the night nurse as a striking diagnostic sign differentiating sound sleep from secondary coma. We have, therefore, introduced a strict rule at Bournewood Hospital that all patients on the insulin coma regimen are to be awakened every three hours and given 100 gm. of glucose in orange juice, in addition, of course, to being watched by the night attendant for excessive perspiration heralding coma. We have not found the addition of glucose during the night to be the cause of any significant interference with the next morning's reaction to insulin except in a few cases during the early phase of the treatment. In such instances, this rule may be modified in exchange for additional measures of strict supervision, still, however, including the precaution of testing the patient's capacity for arousal at the prescribed interval.

ETHER DRIP INFUSION TREATMENT

The dangers of ether drip infusion treatment are local phlebitis and sclerosis of the veins due to excessively high concentration of the ether at the infusion site, or, presumably due to still higher concentrations, fatal pulmonary fat embolism (Lichtenstein and Sewell¹³⁷).

Since it is obvious that both these dangers are due to excessively high concentrations of ether, the important preventive measures are: to see to it that the proper mixture of the infusion fluid is maintained by frequent shaking of the infusion bottle at all times, and, particularly, not to allow the last 50 cc. of the infusion fluid to enter the vein at all, since some excess of ether is bound to accumulate at the top of the column of infusion fluid. This is inevitable even if the bottle has been well chilled and mixed prior to infusion and has been kept well stirred up by frequent shaking throughout the treatment. A mechanical agitator may be desirable, although not absolutely essential as long as an attendant shakes the fluid frequently.

PSYCHOSURGERY

As in all surgery, foremost in importance but fortunately not in incidence among the complications of psychosurgery are operative and post-operative hemorrhages. It is imperative, therefore, that anyone attempting lobotomy should have the facilities as well as the skill to control hemorrhage when it occurs. Sjöqvist and his coworkers¹⁴⁰ and Freeman and Watts¹³⁹ have called attention to a useful means of diagnosis of post-operative hemorrhage which places practical importance upon their procedure of injecting contrast medium into the plane of section, originally intended merely as a method of verifying the level of the transection of the frontal lobe. This plane of section in roentgenographic lateral view usually

appears as a straight line. When postoperative hemorrhage occurs, this line will tend to bulge in a posterior direction.

The danger of infection has been greatly reduced since the advent of the antibiotics. Freeman and Watts¹³⁹ recommend routinely giving penicillin as a prophylactic measure. Three hundred thousand units is injected daily for three days. If signs of infection develop at a later period the dosage is stepped up to 1,000,000 units every three hours; in addition, sulfadiazine is administered until the fever subsides or the blood level reaches 7 to 10 mg. per 100 cc.

Sections that are too far anterior, especially at the base, will sometimes elicit rage reactions or other excited psychotic behavior. Whether Walker's area 13 is implicated in this complication, as Fulton suspects, is not certain. I have seen a case in which active excited psychotic behavior was produced in a woman who was operated on for an involutional depression 40 mm. anteriorly to the usual standard plane as determined by the line drawn from the coronal suture to the sphenoid ridge. After this operation this woman became rather agitated, mistook her daughter for a sister who had died long ago, and developed numerous other disturbing hallucinations, such as seeing dead people walk about again. This behavior did not subside for 42 days. When a second operation was carried out in a plane 30 mm. behind the preceding one, the disturbance subsided and the patient made an uneventful recovery.

The opposite undesirable effect, namely, profound and lasting inertia and apathy, may be produced by cuts that were placed too far posterior. This contingency is difficult to avoid because in schizophrenic patients particularly—as Freeman and Watts¹³⁹ have pointed out—relief is frequently insufficient if the posterior parts of the frontal lobe are not encroached upon. Freeman and Watts have demonstrated in a number of cases that patients unrelieved by what they call their standard lobotomy, carried out from the coronal suture down to the sphenoid ridge, will be relieved when later operated on 10 to 15 mm. behind this line. This, of course, exposes the patient to the danger of producing excessive apathy and inertia. I am certain that Denny-Brown¹³⁸ is right when he assumes that the posterior operation does not remove any fiber systems other than those dealt with anteriorly but merely increases the number of fibers transected. This will be readily apparent by comparing Figs. 43 and 44 (pp. 138, 139). In Fig. 43, which represents a section anterior to the genu of the corpus callosum, the only part of the cingulum exposed in the section at this level is a tangential section of its anterior curve. In a farther posterior plane (Fig. 44) (their relative position in the skull is indicated in Fig. 42), the cingulum is thoroughly and completely transected twice, once above the corpus callosum and another time below. The same increment in posterior direction applies also to the thalamofrontal tract. A posterior cut is bound to transect more fibers than a cut located further anteriorly, because there a good many fibers will already have left the tract, having fanned out to their respective cortical areas on their way from the thalamus forward and will not be cut unless the peripheral convolutional white matter of the individual convolutions to which these fibers go is carefully dissected out. Based on Denny-Brown's impression of the mainly quantitative aspect of the differences between anterior and posterior lobotomies, I would feel that it may be wise to develop more complete dissections anteriorly. It would be of interest to

see whether the results in cases of failure with routine lobotomy may be improved by merely dissecting out the fiber systems more completely in an anterior plane.

If the incision in the upper half of the frontal hemisphere is carried out more than 15 mm. behind the coronal suture, where it is bound to interfere with area 6, the operation permanently interferes with motor skills in addition to sphincter control, thus producing permanent incontinence as well as other forms of frontal dyspraxia. If the cut goes too far posteriorly in the lower half of the frontal lobe, edema and trophic disturbances of the limbs may develop. A still more critical danger of posterior section of the lower halves of the frontal lobes is that of producing pulmonary collapse and atelectasis. Freeman and Watts believe that the respiratory insufficiency leading to pulmonary collapse and atelectasis is part of an excessively profound general inertia, although it is certainly conceivable that it may be due to interference with the areas at the posterior extremity of the orbital lobe which were found to have a profound influence upon respiration. One of Freeman and Watts' patients developed acute pulmonary edema; after improving temporarily and holding her own for fifteen days after the operation, autopsy revealed in addition a pulmonary infarct in the right lower lobe. Freeman and Watts were successful in some cases, one of which they reported at length (Case 162, p. 209), in overcoming postoperative pulmonary collapse by energetic stimulant measures. They sat the patient up, pounding him vigorously on the back and chest, until he coughed; also manipulation of the larynx kept his respiration stimulated. Freeman and Watts found that in the later stages of the condition they could overcome inertia and respiratory inadequacy by the use of benzedrine sulfate. Ten milligrams once or twice a day was found helpful and patients were found to be able to tolerate 50 mg. daily without discomfort. Dr. H. Hamlin and I had an experience of losing a patient from obviously inadequate and disturbed respiratory activity following a second operation which was placed 1.5 cm. posteriorly to the first, the first having been carried out in the standard plane. I wonder whether the use of a respirator may not be helpful in some of these cases. The danger certainly exists that one may fail to take the respiratory difficulty seriously enough in some borderline patients who may later succumb to pulmonary complications.

Convulsions after lobotomy may occur in 4 to 12 per cent of the cases. Usually, however, this does not lead to epilepsy in the sense of a disabling disease. In most cases only one or a few convulsions occur. If they are persistent, anticonvulsant medication is necessary to control them. The incidence of convulsions is higher—25 per cent—after cortical operations such as gyrectomy or topectomy. Following thalamotomy epileptiform convulsions have so far not been observed. The electroencephalograms recorded by Spiegel and his coworkers¹⁴¹ showed no changes or only slight transitory slowing, particularly in the frontal leads.

Last but not least, we must realize that even psychotherapy has its risks and complications in mental disease, foremost among which is the suicidal risk (Oliven,⁹⁷ Farrar³⁵). In view of the fact that approximately 16,000 suicides occur annually in this country, and an additional estimated 100,000 suicidal attempts are made each year, the physician must make a careful evaluation of the risk in order to apply preventive measures and bring the patient promptly to treatment.

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XI

THE QUESTION OF BRAIN DAMAGE FROM ELECTRIC TREATMENT AND INSULIN COMA TREATMENT

The question of brain damage in electroshock therapy and in insulin coma treatment is indeed an important one, since this issue must determine our attitude toward the treatment consciously and unconsciously. While it is obvious that brain damage can occur in conditions which interfere markedly with respiration, circulation, and metabolism, and while it has been demonstrated that brain changes must occur if this interference is excessive, it is by no means a foregone conclusion that such changes are inevitable.

The brain is protected from injury by a number of intricate safeguards, most of them regulated by the sympatho-adrenal system, others inherent in the chemical constitution of the nerve cell itself, e.g., its ability to live for a limited time on its own glycogen stores during the state of hypoglycemia. Sufficient reliable information has accumulated in the past twenty years to allow us to give fairly clear-cut answers to the following questions:

1. Is brain damage inevitable?
2. If so, is it a necessary link in the chain of events leading to therapeutic benefit?
3. Or does brain damage constitute an undesirable and avoidable complication of the treatment?
4. If so, is the result of treatment due to factors other than brain damage?
5. What role do reversible brain changes play in the process of electroshock or insulin coma treatment?

ELECTROSHOCK

The literature is by no means unanimous concerning these points and considerable emotional bias is prevalent in spite of the objective nature of the point at issue. There is also a good deal of uncritical evaluation of morphologic changes, especially of the glia. The glia has functions comparable to those of the reticuloendothelial system; that is, it has functions of an essentially protective nature. Progressive changes of the glia, therefore, should not be interpreted glibly as indicating brain damage, but rather as an expression of the means the body may use to prevent brain damage. Such progressive changes of the glia occur in a variety of generalized infections, including scarlet fever, pneumonia, etc. and the like, in which

there were no other signs of alterations of the brain tissue (Spielmeyer⁵⁰). Such changes also occur in cerebral edema, and are, of course, reversible. Nevertheless, such progressive changes of the glia have been described in the literature (Waggoner and Scharenberg⁵⁷) as proving brain damage caused directly by electroshock treatment, although these authors obtained this finding in a patient who died under conditions suggestive of pul-

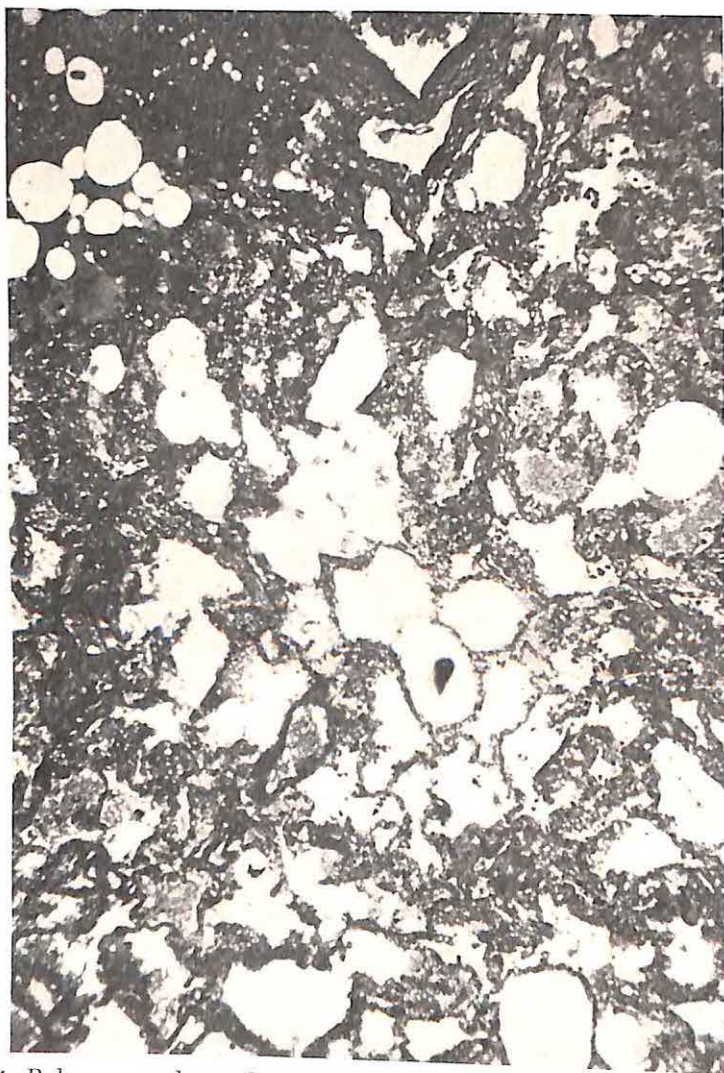


Fig. 46. Pulmonary edema. Section of lung stained with hematoxylin-eosin (x67). Female aged 40, accidental electrocution by low tension alternating current (60 cycles, 120 volts, 300 milliamperes) from left hand to body sustained by turning off ultra-violet lamp while sitting in bathtub. Found dead one-half hour after accident. (This and the following three figures are from L. Alexander, *J. Indust. Hyg. & Toxicol.*, vol. 20, 1938.)

monary edema, which occurred as a complication of an electric treatment of the electric coma variety. It is important in this connection to keep firmly in mind that pulmonary and cerebral edema (Figs. 46 and 47) occur as complications of accidental electrical injuries, in which no current has passed through the brain at all but merely through the heart region,

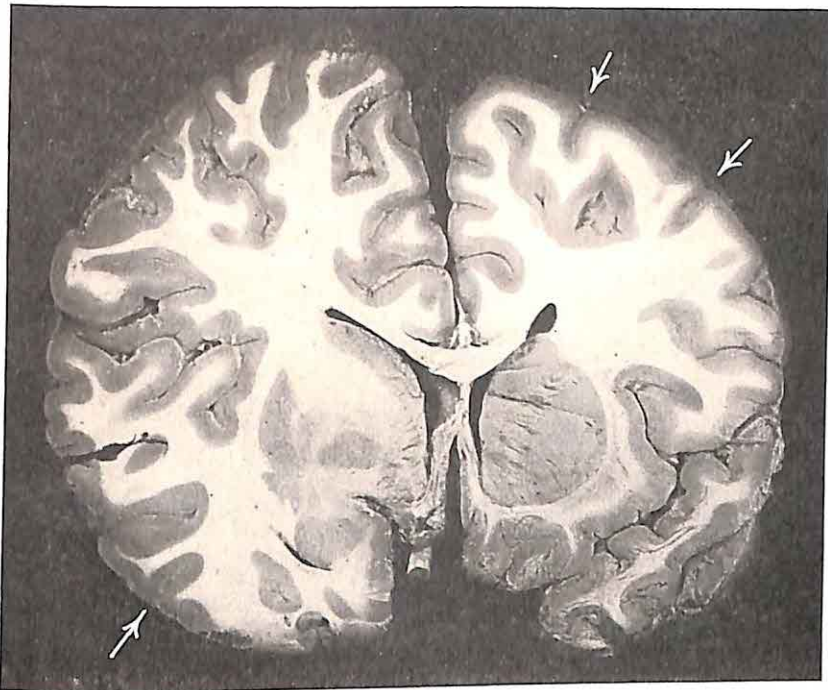


Fig. 47. Cerebral edema. Frontal section through brain at the level of anterior horns of lateral ventricles. Note severe edema of central and convolutional white matter of hemispheres leading to obliteration of the clefts of subarachnoid space normally found in the valleys between adjacent convolutions (especially at sites marked by arrows). Male aged 17. Accidental electrocution by low tension alternating current (60 cycles, 120 volts, 200 milliamperes) flowing from lumbar region to shoulder, sustained from poorly insulated wet heating pad.



Fig. 48. Swollen macroglial astrocyte from the edematous white matter of the brain shown in Fig. 47. Note rounding of contour and granular appearance of upper part of cell body. Cajal's gold sublimate stain (x1250).

and that progressive changes of the glia (Figs. 48 and 49) are a characteristic feature of the brain in such cases. Since pulmonary and cerebral edema are only rare complications of therapeutic electroshock, we certainly cannot generalize from such findings that either brain damage or mobilization of the glia is a regular feature of therapeutic electroshock.

The entire question of pathologic brain changes resulting from experimental electric or metrazol convulsive shock seems to depend largely upon the techniques employed by the various investigators. Reports in the litera-

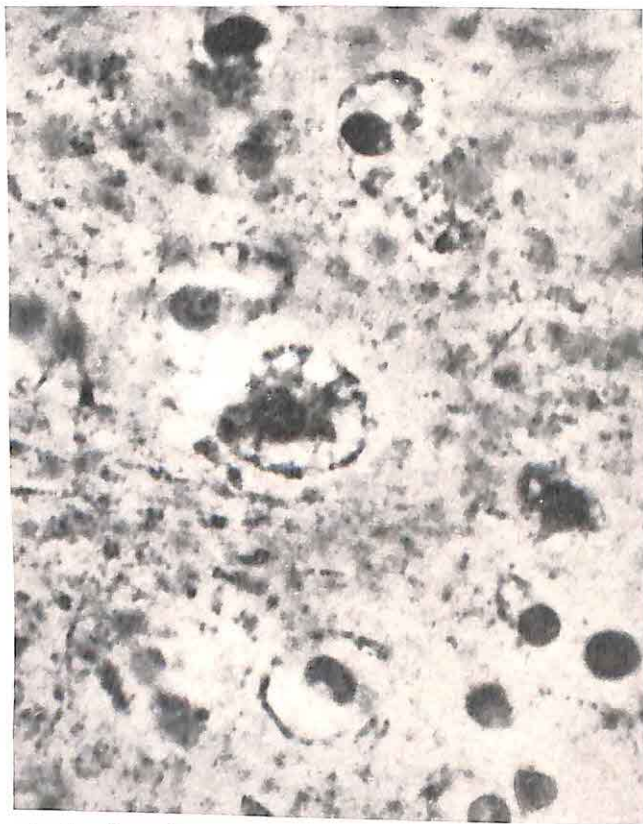


Fig. 49. Oligodendroglia cells, showing various degrees of swelling. From the edematous white matter of the brain of the patient whose lung is shown in Fig. 46. Stain: Hortega-Penfield (x1250).

ture reveal a marked variation in opinion as to the degree or the presence of demonstrable brain lesions, even under similar experimental conditions. Thus, Strecker et al.⁵³ noted variable results in the brains of monkeys subjected to one to fifteen metrazol convulsions; 3 of these animals demonstrated no gross abnormal changes, while others showed subarachnoid hemorrhages extending over the cortex and the base of the brain. In all the experimental brains, vacuolization of the nuclei and loss of Nissl substance were noted. Liebert and Weil³² reported similar findings with metrazol experiments upon rabbits. Two of the group showed no pathologic abnormalities while in others, scattered subarachnoid hemorrhages, perivascular petechiae, mild hypertrophy and hyperplasia of all three types of glia elements were demonstrated together with more or less pronounced

cell shrinkage. Winkelman and Moore,⁶⁰ on the other hand, were unable to discover any pathologic changes in the cerebral cortex of cats killed with a single massive dose of metrazol as compared with sacrificed control animals, aside from a minimal degree of pyknosis of scattered cells, which they felt was within the normal realm. Arieti⁶ reported mild nerve cell alterations in the brains of 3 of 5 monkeys treated experimentally with metrazol and sacrificed with a large terminal dose, but found no evidence of hemorrhage, and only an occasional small area of cells showing Nissl's severe type of degeneration. Petechial hemorrhages in the central nervous system, chromatolysis, cytolysis of neurocytes, pyknosis of ganglion cells in the cortex, and ischemic homogenizing cell changes were observed in the brains of cats treated with metrazol by Neumann et al.⁴⁰ However, identical changes were found in the brains of the control animals, and no clear-cut changes could be found to differentiate the shock-treated group from the control group.

A study of biopsies of the brain following artificially produced convulsions in rabbits made by Heilbrunn and Liebert²³ with a variety of convulsive agents was one of the first experiments to indicate the reversible nature of the majority of the changes observed. These investigators noted the following qualitatively similar morphologic alterations immediately after convulsions induced by insulin, metrazol, electric shock, nitrogen inhalation, air emboli, and monobromidated camphor: loss of sharp outline of ganglion cells, disappearance of Nissl bodies and swelling of cells, some vacuolization, slight swelling of astrocytes and oligodendrocytes. However, thirty minutes after the convulsion these morphologic alterations had markedly decreased in severity, and forty-five minutes after the convulsion no definite changes could be demonstrated in many of the experimental animals. Moreover, the same alterations were observed in biopsies performed after the administration of subconvulsive doses. These investigators concluded that the changes are attributable to anoxia to which the direct toxic effect of the convulsant may contribute, for when the convulsant action of the metrazol was inhibited by the administration of curare, similar alterations were observed.

However, in further experiments the following year, Heilbrunn and Weil²⁴ observed marked histopathologic changes in rabbits treated with electric shock. These animals received $\frac{3}{10}$ to $\frac{1}{2}$ second current passage of 50 to 60 cycle alternating current at 65 to 300 milliamperes, and reacted with convulsions lasting 10 to 30 seconds. One-half the group developed complete paralysis of the hind legs, bladder, rectum, and lower abdominal musculature. Treatments were, nevertheless, continued, in the face of loss of appetite and the development of infections of the urinary tract. Necropsies revealed localized hemorrhages into the pia-arachnoid, cerebellum, and spinal cord, which the authors attributed to the mechanism of the convulsion with its sudden rise of arterial blood pressure, followed by vasospasms of the peripheral arteries and stasis in the venous system, further aggravated by cessation of respiration for several seconds. Similar lesions were noted in the brains of cats subjected to electroshock by Alpers and Hughes.⁴ These animals received up to 23 treatments, some with 60 cycle alternating current, 150 to 200 milliamperes, and others with less high currents. Duration of the passage of the current was not stated; current density was undetermined. A degree of hyperemia in the meninges, subarachnoid and

punctate hemorrhages were observed even in the animals treated with the smallest currents used by them. Moreover, Neubuerger et al.³⁹ found widespread pathologic changes in the brains of dogs given 2 to 25 treatments with 80 volts, 200 milliamperes for 0.15 second, including tigrolysis, paleness, swelling, vacuolization, and in some cases, ischemic and severe changes of the nerve cells, as well as vascular dilatation, minute hemorrhages in the cortex, meninges and around the ventricles in some brains.

The heuristic value of the findings in this and the preceding experiments by Alpers and Hughes and Heilbrunn and Weil was promptly challenged by several investigators. Moore³⁷ suggested that excessive milliamperage is capable of producing disruptive changes such as were noted by Alpers and Hughes; and Ferraro¹¹ pointed out that the duration of the passage of the current may be the crucial factor in determining the presence or absence of such changes. Furthermore, Hassin²¹ stressed that no hemorrhages have been observed in the brain tissues or meninges of humans who have been electrocuted. Hiller²⁵ attributed the changes demonstrated by Heilbrunn and Weil to circulatory disturbances. Neymann⁴¹ pointed out that the electrodes used by these authors in conducting their experiments on rabbits would correspond, if equated proportionally for human use, to electrodes covering both sides of the head and most of the neck, while the strength of the currents employed was severe enough in his opinion to have evoked a convulsive response in a human subject. In addition, Moore³⁸ raised the question that the animals employed by Alpers and Hughes⁴ may have suffered from nutritional deprivation and that their vasculature, possibly predisposed to weakness by vitamin deficiency, was additionally disrupted by the electric shocks and gave way, resulting in hemorrhages. Kalinowsky²⁸ raised still another issue in this connection by mention of the work of Stender,⁵¹ who observed hemorrhages of the same type in animals following metrazol convulsions and was impressed by their similarity to those seen in cases of concussion. He therefore conducted a second series of identical experiments, however, confining the animals within a padded box during the convulsive period, and was unable to demonstrate evidence of a single hemorrhage in this group.

In the meantime, evidence gradually accumulated demonstrating that a good many of the changes noted in the brains of experimental animals treated with electroshock either were transitory reversible changes or were due to inadequacies of the technique, such as passage of excessive currents through the vasomotor centers, or to other extraneous factors such as trauma, or even to artefacts caused by postmortem changes. Barrera et al.⁸ published an important study in which monkeys received electroshock treatment series in a careful duplication of the human treatment setting. Comparisons of these brains with those of control animals revealed that similar changes were to be found in both groups, and no unequivocal histopathologic changes were manifested in the shock-treated group which were not also found in the control group. Moreover, neuropathologic findings were surprisingly meager, even in the brains of animals subjected to a treatment series beyond the clinical level, and these investigators were unable to point out any quantitative relation of brain changes to the frequency, number of treatments, voltage, or convulsive response. Electroencephalographic changes were, however, definitely similar to such changes found in human subjects treated with electroshock, and constituted, the

authors felt, definite physiologic evidence of transitory reversible dysfunction.

In the same year, Echlin,¹⁰ in experiments using cats, succeeded in demonstrating that temporary areas of focal cerebral ischemia can be produced by electrical or mechanical stimulation of the pial arterial vessels, which causes vasoconstriction of these vessels. The pial vessels of dogs and monkeys were found to be somewhat more resistant to this type of stimulation. Using the intravascular perfusion method of fixing the brain tissues of a monkey which had received ten experimental electroshock treatments in a clinical treatment setting, Fetterman¹⁴ reported absence of lesions, cell destruction, glial reactions, and cellular changes in any of the brain sections. Globus et al.,¹⁸ employing the electronarcosis treatment on dogs, investigated the effects of the application of 200 to 700 milliamperes for 30 seconds, followed by a decrease of current to a level commensurate with unimpaired respiration (60 to 140 milliamperes) and maintained for from 15 to 75 minutes. No pathologic alterations of any sort were noted. Moreover, some of these experimental animals were sacrificed at the end of their treatment series by electrocution, and it is significant that the histologic structure of these brains did not differ either from those of the other experimental animals killed at the termination of the treatment series by ether injection into the lungs, or from those of the control animals. Likewise, Lidbeck³¹ found only minimal pathologic changes in the brains of dogs who had received electroshock treatments in a careful duplication of the human treatment situation. These consisted chiefly in some shrinkage and ischemia of ganglion cells near the site of the electrodes.

A subsequent experiment was conducted in 1943 by Heilbrunn²² aimed at the prevention of hemorrhages in the brain in experimental electric shock. He concluded that preliminary injections in rats of atropine sulfate, synthetic vitamin K, calcium gluconate, and a thromboplastic suspension of brain substance as well as various combinations of these preparations were of no value in the prevention of hemorrhages in the brain. These animals were treated with an alternating current of from 10 to 20 volts with a strength of from 30 to 50 milliamperes for $\frac{3}{10}$ second, through temporoparietal regions. However, those rats in which convulsions were inhibited by preceding ether narcosis displayed no hemorrhages, and the author concludes that the causative factors in the production of these lesions are changes in pressure accompanying the convulsion, rather than the passage of the current itself. Winkelman and Moore,⁶¹ on the other hand, emphasized that when hemorrhages are found in the brain of experimental animals following electrically induced convulsions, one or more of three factors must have been operative: (1) the intensity of the current was excessive in comparison with human therapeutic range; (2) during the course of the experiments the animals were nutritionally under par and suffered from vitamin deficiencies; or (3) the animals sustained mechanical craniocerebral damage, resulting from inadequate protection or restraint during the convulsions.

More recent experiments conducted by Windle et al.⁵⁹ corroborate the importance of this third factor; whereas chromatolysis and nerve cell degeneration were shown to be consistent features of the concussion syndrome in guinea pigs, similar experiments made it clear that the massive discharge of neurons with electric shock does not produce the same effects.

In these experiments, one group of guinea pigs received the minimum shock required to produce a convulsion, while a second group was subjected to 100 volts, 650-725 milliamperes, for 6 to 12 seconds, in an attempt to produce maximal damage. The perfusion method of fixation was used, together with thionine staining technique. This method, when promptly employed under controlled hydrostatic and osmotic conditions, eliminates the autolytic changes which can so easily distort the picture of normal brain structures. Use of the buffered thionine technique eliminates the need for overstaining and decolorization staining procedures; since the thionine method will adequately stain the glial cells, special procedure for examining these cells can be eliminated. When, however, the more common immersion-fixation method is employed, a gradient of fixation results from surface layers inward. As Siekert⁴⁶ points out, "failure to recognize postmortem artefacts may explain much of the cytopathologic alterations reported by other investigators who have attributed their observations to electric shock." In the study reported by Windle et al.⁵⁹ the examining histologist was kept ignorant of which brains were those of control animals, and which were those of shock-treated animals. No edema, no changes in distribution or number of neuroglia, no structural alterations of nerve cells in any part of the brain could be demonstrated in either of the groups. Windle and his coworkers stress what they feel to be the root of the variability of reported results in experimental pathology: the amount of time elapsing between death and fixation of tissues, the manner in which the tissues are fixed, the type of stain employed, and the degree of care with which the tissues are preserved. It is extremely interesting to note in this connection the results of studies of the cervical enlargement of the spinal cord of guinea pigs made by Koenig²⁹ employing thionine stain exclusively. One-half hour after death, perceptible changes in the larger neurons and early fragmentation of the Nissl bodies were noted in the tissues of normal control animals. These changes were progressively more striking at greater intervals of time. By twenty-four hours after death, little suggestion of discrete Nissl bodies remained discernible; the nuclei also showed changes, becoming indistinct.

A study made by Alexander and Löwenbach² in 1944 demonstrated that the significant morphologically recognizable tissue reactions seen from a few minutes to nine days after electroshock treatments in cats were limited to the parts of the brain which lay in the direct path of the current; that is, the frontocruciate lobes. An earlier study by Alexander and Weeks³ had pointed out that the electrical current passes through the animal body as if it were passing through a structureless gel, always choosing the shortest path from contact to contact without deflection by anatomic landmarks. Single shock treatments in a treatment setting similar to the human one produced no demonstrable changes of the neural parenchyma, four minutes to seven days after administration of the treatments. Reversible arteriolar vasoconstriction was the only alteration found to be present shortly after administration of treatment but was consistently absent upward of thirty minutes after treatment.

Ferraro et al. in 1946,¹² and Ferraro and Roizin¹³ in 1949, carrying out experiments in monkeys also concluded that most of the changes observed were of the reversible type. Many of the changes noted were related to circulatory disturbances and to increased permeability of the blood vessel

walls. Histologic alterations were more pronounced and more frequent in areas traversed by the direct path of the current; moreover, the severity of the alterations observed was proportional to the intensity of the current, the duration of the current flow, and to a lesser extent, to the number of treatments given (these animals received as many as 100 treatments). The importance not only of the milliamperage employed, but of the relative size and placement of the electrodes and the current density per square millimeter of the diameter of the path, had been found to be crucial factors in the production of reversible or irreversible brain changes by Alexander and Löwenbach.² An important contributing factor, involvement of the hindbrain through excessive stimulation of vago-vasomotor centers of the medulla oblongata by the current, in turn creating cardio-circulatory disturbances which can interfere with the integrity of the brain tissue, can be overcome by applying the electrodes far enough forward. However, cardio-circulatory disturbances can also be caused by the strain of excessive demands on these systems made by the mechanism of the convulsion. This can be eliminated in animal experiments by careful selection of only healthy animals. It is this factor which appears to be directly responsible for the types of changes described in human brains. Human brain changes reported in the literature (see pp. 170ff., references 3, 30, 34, 54, 66, 85, 86, 94, 108, 109) as having resulted from electroshock treatments seem to be representative of those found in the brains of individuals who have been exposed to incidents of circulatory-respiratory embarrassment due to a variety of causes, such as coronary heart disease, asphyxia under anesthesia or incidental to ingestion of other toxic substances, or following episodes of collapse due to physical exertion in persons with cardiovascular disease. Such changes would undoubtedly have been evident on autopsy after any excessive cardio-circulatory or asphyxial stress, even without electroshock. These findings teach us what precautions to take, both in the selection of patients and in the administration of therapy, but they are not representative of those organic changes which must take place routinely with electroshock.

Although Freeman and Watts¹⁶ favor the view of regarding shock treatments as resulting in "concealed destruction of the nervous system" (page 383) producing a "disseminated or diffused" instead of a "localized lobotomy" (page 386), they nevertheless failed to demonstrate, in the numerous biopsies and postmortem specimens from lobotomized patients that they studied, any significant cell alterations of the cortex. Their cortical biopsies, including one which was taken the day after four successive electroshock treatments had been administered (page 407), were consistently free from pathologic changes.

Distinct physicochemical changes do take place, however, following electrically induced convulsions, the nature of which provide a fruitful area for further research. Spiegel-Adolf et al.⁴⁸ have demonstrated that, in dogs treated with electroshock, the electrolyte content of the cerebrospinal fluid is increased, and a distinct increase in potassium and phosphate contents also takes place. Electrical stimulation of the brain appears to effect a brief release of ions from the cells of the central nervous system; non-electrolytes in the spinal fluid also increase following a convulsion, and do so more markedly than do the electrolytes. The authors feel that this increase is not attributable either to the increased permeability of the blood-liquor barrier, or to abnormal content in glucose and proteins, but

that a mobilization of nuclear substances—presumably due to breakdown of nuclear substances—takes place under the influence of convulsive treatments.

Spiegel-Adolf, Wilcox, and Spiegel⁴⁹ confirmed these findings for humans under electroshock treatment, and made the additional discovery that not only nucleic acids and their constituents increased in the spinal fluid, but related enzymatic substances, namely nucleases and desaminases, increased as well. This, of course, is by no means necessarily indicative of chemical brain cell damage, as it may suggest at first glance. Spiegel and his coworkers⁴⁹ feel that this observation may indicate that electroshock increases the activities of enzymes related to the nucleic acid metabolism of the brain, which may be presumed to be deficient in view of the lack of enzymes and coenzymes in mental diseases, particularly schizophrenia, demonstrated by Hoskins²⁶ studies.

Summary. In conclusion as to the question of brain changes in electroshock, we can answer as follows:

1. Brain damage in electroshock treatment is not inevitable.
2. Brain damage therefore cannot be regarded as part of the mechanism by which therapeutic electroshock accomplishes its results.
3. Brain damage constitutes an undesirable complication due to profound respiratory and circulatory disturbances that may result from electroshock therapy, either due to relatively excessive strength of current, technical imperfections such as failure to restore respiration promptly or to supply an unimpeded airway, or to unwise treatment of individuals with diseased or unstable cardiovascular apparatus. Within reasonable limitations these complications are avoidable by safeguards based on an understanding of the physiology of the procedure (see Chapters X and XIV).
4. The result of electroshock treatment must be due to factors other than brain damage.
5. The role of possible reversible brain changes is not yet adequately understood. There is suggestive evidence of changes in the enzyme systems of nucleic acid metabolism (nucleases and desaminases) of the brain cells.

INSULIN

Animal experimentation provides the most valuable tool for the histologic evaluation of the effect of insulin on the brain and other organs. The tissue can be fixed immediately after death, thus avoiding the pitfalls of postmortem changes. The comparative effects of minimal and maximal doses, light and deep comas, interrupted and uninterrupted hypoglycemia, mild and severe convulsions can be studied and analogies drawn with control preparations. Human material, on the other hand, is (fortunately) scarce; moreover, it is variable. Tissues are usually not fixed for examination until enough time has elapsed for important and misleading post-mortem changes to take place, and in the case of the human subject it is impossible to determine what previous degree of injury or change has been produced by illness or other physical abnormalities.

Particularly the study of the brains of diabetics who have died in hypoglycemia cannot be considered seriously in a study of the effects of insulin on the nerve cells since a different set of factors complicates these cases.

Study of the brains of schizophrenic patients who died in insulin coma

treatment, while helpful in demonstrating the severe changes that can be produced by complications such as irreversible coma and circulatory and respiratory accidents, does not tell us whether or not brain damage is a regular side effect of insulin coma treatment, or, as some contend, the essential feature of the treatment, or whether it is merely a complication of the treatment which is avoidable by proper management of the coma.

For these reasons, our conclusions concerning the role of brain damage in insulin coma treatment will have to be based mainly on the results of animal experimentation in a controlled situation. Even here we are faced with the fact that few scientists have attempted to reproduce the standard insulin coma treatment situation, for the most part contenting themselves with experiments directed toward producing maximal irreversible brain changes either by use of massive lethal doses, or prolonged and severe convulsions, or by permitting the animals to die either in hypoglycemia or secondary comas. Even in such cases,^{1, 15, 43, 45, 52, 54, 55, 56, 60, 62, 63, 64} a remarkable nonuniformity of results was obtained.

Some of the variability of the brain changes reported can be attributed to the fact that different species of animals were employed. As Lorentzen³⁴ points out, those experimenters who used dogs, cats, or monkeys demonstrated the most severe acute degenerations, i.e., chromatolytic cellular changes, because these animals, with a more differentiated central nervous system, are more resistant to hypoglycemic shocks than rabbits or guinea pigs, and as a consequence, "the histologic changes may reach a more conspicuous degree of development before the animal dies." Thus Stief and Tokay,⁵² upon subjecting dogs to repeated insulin comas extending over a full day without interruption and complicated by severe and prolonged convulsions, during which the animals either died spontaneously or were killed when they reached a moribund state, found changes of an ischemic and anoxic nature, including severe and pyknomorphic ganglion cell disease. The glia remained inactive in some cases, in others, oligodendrioglia and microglial reactions were seen. Schereschewsky et al.⁴³ also directed their experimentation upon dogs toward producing maximally severe lesions with massive doses and uninterrupted comas. They reported cloudy swelling of the liver and kidney, acute degenerative atrophic changes of the ganglion cells, edema, hyperemia and hemorrhagic extravasations in the brain and spinal cord, necrobiotic changes in the adrenal glands consisting in a decrease of lipoid content and destructive cell changes. They regarded insulin in large doses as a "sympathotropic vascular toxin." Accornero,¹ on the other hand, reported that in his investigations of dogs allowed to die in coma, only part of the group displayed irreversible changes such as hyperchromatic nuclei, perinuclear clear zones, vacuolization of the ganglion cells, progressive changes as well as pyknosis of the glia, areas of necrotic destruction, and so on. A large number of the group sacrificed in coma showed a reversible type of Nissl's acute cell change. However, Winkelman and Moore⁶⁰ found that the brains of cats which died in secondary hypoglycemia, after having been subjected to deep coma and convulsions, showed all stages of cell degeneration, homogenization, vacuolization, necrosis, and poor staining. Finley and Brenner,¹⁵ working with monkeys, reported that acute nerve cell change, probably of a reversible nature, is evident in the brains of monkeys kept in coma three and one-half hours, while upward of nine hours of coma

produces irreversible changes with dropping out of nerve cells in the cortex and acute glial reaction. Similar irreversible changes were reported by Dünner et al.,⁹ working with rabbits. These investigators found, in addition to degenerative cell changes, vascular disease with endothelial and adventitial proliferation, similar to Wernicke's disease, irreparable parenchymal destruction, retinal hemorrhages, choking and atrophy of the optic disks.

Tannenbergs,⁵⁴ in a study of the comparative changes produced by anoxic and insulin shock on rabbits, noted that the anatomic changes were the same in both types of shock, i.e., degeneration of the ganglion cells and reactive glial proliferation. However, a marked difference was demonstrated in the morphologic appearance of the ganglion cells; in insulin, a fading away of the cells took place, leaving "bleached areas" in the stained preparations, while in anoxia the cells appeared shrunken and darkly stained, as if calcified. Baker and Lufkin⁷ produced severe hypoglycemic convulsions in rabbits with intravenous injections of insulin; the convulsions were allowed to continue for varying periods up to one hour, and the treatment was then terminated with dextrose. All the rabbits revived instantly, and appeared normal in the interval between treatments. They were sacrificed five days following the last treatment, and to the surprise of the investigators these brains showed very little change. No striking cell lesions were noted. All cell elements were intact. An occasional petechia was seen, and some extravasations of erythrocytes within the meninges. Two animals were allowed to die in coma; immediate necropsy showed mild but by no means prominent chromatolysis, involving only a few scattered groups of cells. There were no cell alterations of pathologic significance.

Similarly, Weil et al.⁵⁸ found only mild changes in the brains of rabbits given insulin doses to the level of the convulsive threshold and then promptly revived with sugar. In the brains of two animals which died during convulsive seizures, some pale staining was observed, and a mild degree of homogenization in the Purkinje cells of the cerebellum, but no pathologic changes of any significance could be found in the cortical neurons, striatum, or thalamus, although many shrunken cells were noted in the Ammon's horn. Vascular changes were mild even when the duration of the treatment was the longest. No proliferation of the capillaries or damage to the vessel walls leading to perivascular hemorrhages was observed in any of the rabbits; nor was there evidence of demyelination or proliferation of mesenchymal tissue. Those rabbits which received the largest doses of insulin displayed the most marked changes, but no similar correlation was found between the number of seizures and the amount of brain damage. These authors suggest that intracellular anoxia during insulin coma treatment may be responsible for severe generalized disease of the neurons following prolonged treatment with large doses of insulin.

On the other hand, Grayzel¹⁹ concluded from his experiments with rabbits that the greater the number, severity, and duration of the convulsions, the more severe were the brain lesions produced. Those rabbits which did not have convulsions showed no brain lesions; those rabbits whose treatment was terminated promptly upon production of convulsions showed no or only slight brain damage; while those rabbits which had severe convulsions one or more times showed definite central nervous

system lesions. Zones containing shrunken hyperchromatic cells with corkscrew processes were considered as the milder lesions, while, in addition, zones of necrobiosis were noted in the more severely damaged brains. Schmid,⁴⁴ in contrast to Weil's findings, demonstrated in rabbits that even in those animals which had been subjected to the greatest number and duration of convulsions, the Ammon's horn was found to be intact. Like Grayzel, Schmid found no significant changes, except perhaps for slight progressive changes of the microglia, in insulin coma. He noted no abnormalities in the cerebral cortex, no marked changes in the medullary regions of the telencephalon, and no accumulation of fatty substances in these cells, such as were reported by Stief and Tokay. This discrepancy, as Schmid points out, can be attributed to the fact that he did not permit his animals to die in coma, but instead attempted to reproduce the human therapeutic situation.

Appel et al.,⁵ in an experimental situation closely paralleling the work of Grayzel,¹⁹ Schmid,⁴⁴ and Weil,⁵⁸ demonstrated on cats that when the hypoglycemic state is terminated promptly upon production of the first convulsion, brain damage is either nil or slight. Nine cats given identical treatment with identical termination procedures showed dissimilar results. Two of the animals showed no changes anywhere in the central nervous system. Five others showed scattered ischemic changes; the Purkinje cells of the cerebellum seemed most susceptible in all the affected cases. Those cats which showed no evidence of damage had had both numerous treatments and numerous convulsions. Yannet^{62, 63} also employed cats in his experiments. Sixty per cent of his animals given massive doses of insulin without additional therapy survived. About half of these showed no demonstrable evidence of involvement of the central nervous system and histologic examination of these brains showed negative findings. However, chemically it was possible to note a change in cellular composition, particularly a loss of intracellular potassium. The remaining animals in which marked cerebral damage was evident also demonstrated a profound loss of cellular water which led to cell shrinkage and increase in extracellular fluid as well as a proportionally greater loss of cellular potassium.

A series of experiments conducted by Tyler and Ziskind^{55, 56, 64} in 1940 were directed toward determining whether dosage, duration of hypoglycemia, or depth of hypoglycemia was the crucial factor in the production of cerebral lesions in cats. Their results seem to show conclusively that no straight line correlation exists between the degree of brain damage and the amount of insulin dosage, although the greater the dosage, the greater the incidence of brain damage. There also was no correlation between the degree of brain damage and the duration of hypoglycemia per se without regard to its degree, or the duration of the coma period per se without regard to its depth. The depth, or phase, of coma was the inalterably decisive factor in determining the extent and the degree of cerebral injury. *Brain damage always occurred when the cat was held in the medullary (maximally deep) phase of coma for one hundred minutes or more.* The time that the animal must be held in the medullary phase to produce damage varies indirectly with the body temperature of the animal. Cats in which the body temperature was artificially maintained at or near normal during hypoglycemia displayed a far greater incidence and degree

of brain damage than "cool" cats; moreover, in the latter group, *no brain damage occurred in up to 45 minutes of medullary phase coma, whereas brain damage was noted in the "warm" cats at upwards of 25 minutes.* From these data, Tyler and Ziskind⁵⁶ concluded that a greater degree of tissue anoxia results when the body temperature is kept at normal levels during insulin shock than when the body temperature is permitted to drop, because of the body's greater need for oxygen at higher temperature. Although the drop in body temperature is less pronounced in humans than in cats, Tyler and Ziskind feel that this point may explain some of the prolonged comas and accidents occurring during insulin coma treatment.

Experiments by Yannet⁶³ on the effect of pentobarbital on cat sensitivity to insulin, and similar experiments performed by Tyler,⁵⁵ have demonstrated that pentobarbital increases sensitivity to insulin dosage. Yannet pointed out that since the anesthetized animals passed into a hypoglycemic state and thence into a moribund state without exhibiting convulsive phenomena, convulsions per se play no role as an etiologic factor in producing brain damage, thus corroborating Baker and Lufkin's⁷ similar conclusions. Tyler and Ziskind, however, found that although sensitivity is heightened, sodium pentobarbital also exerts a certain protecting influence, and that 150 minutes of medullary phase coma were necessary to produce irreversible injury in such cats.

In evaluating the brain changes reported above, it is important to bear in mind, as Lorentzen's³⁴ careful analysis points out, that the imperfection of staining techniques with aniline dyes has previously rendered the assessment of experimental data extremely difficult. This is particularly true when conclusions are drawn as to changes of the ganglion cells. The evaluation of glial change is further complicated by the fact that in rabbit brains, encephalitic changes, which may easily be misconstrued as the direct result of experimental interference, are frequently in evidence in the form of an increase in the glia and glial hyperplasia. These facts could well account for the variety of results reported by Stief and Tokay,⁵² Tannenbergs,⁵⁴ and Baker and Lufkin,⁷ the two former experiments disclosing proliferation, while the latter investigators demonstrated no glial changes. Similarly, the results of Winkelman and Moore's⁶⁰ investigations are contrasted with the absence of changes demonstrated by Appel et al.⁶

Lorentzen's³⁴ basis for the evaluation of cellular changes is based on Einarson's findings (as quoted in Lorentzen) on the histochemical correlates of cellular activity, as observable in preparations stained with galloxyaniline. Einarson distinguished three stages: (1) the chromoneutral condition during which the cells are at rest or in a normal state of activity; (2) chromophily, characteristic of a beginning increase of activity over the norm; and (3) chromophoby which characterized cells that were kept in a state of increased activity over prolonged periods. Increasing chromophoby is an indication of increasing cell fatigue and exhaustion; extreme chromophily may characterize the acute inhibition, decrease, or abolition of cell activity.

Proceeding with these definitions, Lorentzen³⁴ demonstrated slight to medium chromophoby in the nerve cells of rabbits which died in their first shock treatment; the glia was swollen. These changes were attributed to hypoxia. In the brains of those animals which were subjected to daily insulin shocks for 6 to 28 days and killed immediately after the last shock,

chromophoby was again noted, in increasing degrees when increasing numbers of treatments were given. However, when animals from the same group were sacrificed seven days after the cessation of treatment, the nerve cells in many areas showed slight chromophily. Furthermore, many intensely chromophilic cells and cells in the process of transition between chromophily and cellular atrophy were noted. Lorentzen concludes from his data that after the cessation of treatment chromatin (re-synthesized nucleoprotein) will again accumulate in the cells, and "in most areas the chromophoby will be replaced by the normal activity picture with chromoneutrality." Some irreversible "dropping out" of neurons will be observed in the cerebral cortex—particularly in the fifth layer of the frontal lobes—in the brains of those experimental animals subjected to repeated, severe, and prolonged insulin shocks. During the recovery phase after cessation of treatment, and also during treatments that have not extended over too long a period, slight chromophily—of a transitory and hence reversible nature—will be observed in the brain stem and spinal cord. Conspicuous glial changes will not be found; but a slight increase of the glia in the deep layers of the cerebral cortex will be noted in the brains of rabbits subjected to particularly severe and prolonged shocks.

It is important to note the paucity of significant irreversible changes found by Lorentzen in his experiments with rabbits, despite the fact that the depth of the insulin coma treatment employed by him was so excessive that 4 out of 9 animals in his first series died in shock and 2 others succumbed later on the same day from the direct effects of the treatment. These animals had been held in coma from 60 to 360 minutes, yet the quality of the cell changes reported was essentially of reversible type. Of a second group of 15 animals subjected to a series of from 6 to 28 treatments, 3 died in coma, and a fourth succumbed in secondary coma; here too the changes reported were of a predominantly reversible nature.

Summary. In summary, we can say that in regard to insulin coma treatment the answers to our fundamental questions are as follows:

1. Brain damage is not inevitable. We are able to draw this conclusion particularly from Tyler and Ziskind's^{55, 56, 64} studies, and by implication from Lorentzen's study.³⁴

2. Brain damage therefore cannot be considered a necessary link in the chain of therapeutic events. This fact is confirmed also by other physiologic and clinical observations; for one thing, changes in the autonomic response pattern are fundamentally different from those produced by known brain damage, including that of frontal lobotomy. The changes of autonomic response pattern produced both by insulin coma treatment and by electroshock treatment differ not only from each other, but also particularly from the changes in autonomic response pattern produced by frontal lobotomy. Following lobotomy the reactions to sympathetic as well as to parasympathetic stimulation become first enhanced and then reduced, the release of responsiveness to sympathetic stimulation predominating in the early phases. In insulin treatment the opposite, namely the toning down of excessive responsiveness to adrenergic stimuli, is the most prominent effect of the treatment (Funkenstein et al., p. 30, ref. 13). I have been able repeatedly to confirm this observation in my own case material. However, in those rare cases in which responsiveness to adrener-

gic stimulation was grossly inhibited before treatment, insulin coma treatment brought about its release. Another important point in this connection is of course that brain damage, when it occurs, is a detriment to the quality of the therapeutic result, rather than an asset.

3. Brain damage constitutes an undesirable and in most cases avoidable complication of insulin coma treatment. It can generally be avoided if the deepest (medullary) phase of coma is limited to a maximum of fifteen minutes (see Tyler and Ziskind), and if irreversible coma or circulatory and respiratory upheavals can be avoided.

4. The therapeutic results of insulin coma treatment must be due to factors other than incidental brain damage.

5. The role of reversible brain changes is not understood. It is probable that rearrangements of nucleoproteins take place, as manifested by Lorentzen's³⁴ chromophobia and reactive chromophilia of the nerve cells. Similar changes were seen by Swedish workers (Hyden,²⁷ Hyden and Hartelius,^{27a} Hartelius²⁰) following malononitrile treatment. Nevertheless, malononitrile has failed to bring about comparable clinical therapeutic results (MacKinnon et al.,³⁵ Meyers et al.³⁶). The possible role of these transitory changes, therefore, remains entirely obscure and constitutes an important area for further research.

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XII

MODE OF ACTION AND RESULTS OF PSYCHOTHERAPY AND OF PHYSICAL TREATMENT OF MENTAL DISORDERS

THE DYNAMICS AND EFFECTS OF PSYCHOTHERAPY

The fundamental dynamic forces in mental disorder are hate, guilt, and fear. It is obvious that the treatment for hate is love (Thompson²⁴), the treatment for guilt is understanding, and the treatment for fear and anxiety is communication (Ruesch and Prestwood²¹). The latter authors devised and carried out an important and well controlled series of observations and experiments concerned with communication and interpersonal management of anxiety. They found the most successful method for handling anxiety consisted in sharing through communication. They concluded that "the process of communication is essential for healthy functioning so that people may combine efforts to cooperate, complement and increase their ability to cope with animate and inanimate surroundings. The successful management of anxiety generated in daily life seems possible only through the process of sharing and communication. Alleviation of anxiety through personal contact is the process which is basic to all interpersonal relations from babyhood to old age."²¹

The power of such communication, which is an important element inherent in psychotherapy, has been evident to the earliest observers, especially if such communication is motivated by love in the widest sense of the word including the religious, and guided by understanding. Under such circumstances it is capable of producing profound psychic and physiologic effects. The power of this effect is most striking in the earliest periods of life. The capacity of a mother to transform an anxious, desperate, kicking, and wailing little creature into a smiling cherub by the sound of her voice and the gentle touch of her arms is an experience known to all. It is this extraordinary receptiveness for and dependence on the mother's warmth and closeness which makes the relation of the child to his mother such a crucial experience in a person's life, determining his basic pattern of interpersonal relationships, the future development of trust or mistrust in himself and others. The reason why in later life this direct ministering to the individual's needs, such as is accomplished by the child's mother in infancy, is not as readily effective is due to the fact that these needs become hedged around by a series of intricate defensive operations which prevent the satisfaction of the same

emotional needs which brought these defenses into being. The building of these defenses occurs as a reaction to the difficulties and especially the frustrations encountered in the quest for gratification of these emotional needs in an earlier phase of life.

An important task of the psychotherapist, therefore, is to struggle with the defensive operations. These defensive operations used to be referred to as repressions in the past, somewhat interchangeably with defenses. Rado^{17a} redefined them as "pain barriers," interposed between the unconscious mental life and awareness, as well as between awareness and communication. Anna Freud⁶ has pointed out with great clarity that certain defenses allow a partial or disguised gratification of the impulses against which the defense is directed, thus not actually repressing the impulse entirely. The word repression, therefore, should be limited to only that type of defense in which there is actually complete repression.

This brings up the important subject of the differences in the value and efficacy of the various forms of defenses.

Types of Defenses. In the following I should like to discuss the major defensive operations, in the approximate order of their effectiveness and participation in normal mental life, progressing by degrees from the effective and normal ones to the ineffective and abnormal ones.

Sublimation is the substitution of a socially acceptable or constructive drive for a socially unacceptable or destructive one, such as the substitution of love for sex, of leadership for the power drive, of scientific discovery for the hunting instinct.

Concentration is the ability to devote one's entire attention to an objective task at hand by excluding other objective and personal interests, particularly those that are irrelevant, inconvenient, or that run counter to the task at hand. This defense breaks down under the all pervading egocentricity of mental disturbance; hence, inability to concentrate is a frequent early complaint in mental disorders.

Identification is the unconscious or conscious patterning of one's self in the image of a parent figure or leader; the ensuing mimesis is a strong factor in the intellectual and ethical development of individuals and in the cultural advance of population groups (Toynbee²⁵).

Displacement is substitution of a permitted target of a thought, wish, urge, or act for a sacrosanct or forbidden one such as, marrying an older woman as a substitute for mother; or being hostile to a waitress instead of to one's wife; or being afraid of a "totem" animal of great physical strength instead of father, such as the horse in the case of Freud's famous case of "Little Hans."

Reaction formation is the transformation of a disturbing urge into an attitude expressing its very opposite, derived from the ideals learned during the latency period (age 10-14): such as the substitution of cleanliness for chaotic sex drives, of kindness for hostility, of reasonableness and considerateness for aspirations of omnipotence. As long as this defense holds it is fairly good protection against mental disease; but it leaves the patient wide open to psychosomatic illness. The difference between reaction formation and real saintliness and cleanliness is the uncompromising compulsive inflexible rigidity of attitudes determined by reaction formation.

Isolation is the successful segregation of a disturbing wish, thought, act,

or fact from the remainder of the mental life by stripping it of its associative connections and thus depriving it of its affective meaning, resulting in "symbolic amnesia" (Freud)⁷ for that particular isolated mental content. This defense is akin to normal concentration but less objective, particularly because of its emphasis on disregarding a particular area of the mental content. The result of the defensive operation of isolation may range from the ability to disregard a toothache, to the ability to transform and encapsulate a deeply disturbing wish into a seemingly meaningless habit or obsessive ritual, such as a desire to masturbate into a compulsive need to slide one's finger over a polished smooth surface.

Denial is the bland disregard either in words, actions, or both, of a disturbing part of reality or the inner mental life and the capacity to act as if this disturbing part of reality or the inner mental life did not exist.

Reversal is the change of a strong desire or urge into actual aversion, or the change of any other drive or feeling into its direct opposite, as a result of an experience of unbearable frustration and enforced renunciation encountered in the quest for the desired satisfaction: such as the disgust for the mother's nipple shown by the completely weaned infant, or the development of coldness and aloofness as a result of rejected warmth and tenderness.

Intellectualization is the transfer of a strong urge or desire into a merely intellectual concept, thus depriving it of its quality of reality and impact upon feeling and action.

Undoing is the performance of an act or acts designed to undo the effects of disturbing wishes, thoughts, facts, or acts. It is akin to the childish practice of "blowing the hurt away." The performance of such symbolic or "magic" acts is an ancient traditional aid in the attempted isolation or denial of a conflict or a stressful situation. Their meaning is easily understood. This type of defense is particularly frequent in obsessive and compulsive neurosis.

Procrastination and repetition are self-explanatory. As defensive operations they are almost pathognomonic of attempts of the ego to counteract the desire to masturbate.

Repression is the total elimination from consciousness of a disturbing wish, drive, thought, or fact as well as the exclusion of the unconscious wish or drive from disguised gratification.

Inhibition (specific). A specific inhibition is an attempt of the ego to prevent the realization and gratification of a strong but disturbing wish, urge, or desire; this inhibition may include also those parts of the motor and sensory apparatus that are designed or equipped to be the effector or receptor organs of the acts or sensations actually or potentially participating in the forbidden and suppressed gratification, such as in hysterical paralysis, aphonia, deafness, or anesthesia.

Anticathexis is a defensive operation aimed at preventing cathexis. Cathexis is the ability to "make emotional investments," to become attached and interested emotionally in people and things. Cathexis comes from the Greek, *κατεχέιν*, meaning to behold, to embrace, in a wider sense to be interested emotionally in, in a figurative sense to embrace, a friend, a belief, a policy, a creed. Anticathexis results in attitudes of coldness and aloofness with scrupulous avoidance of any emotional entanglements.

Ego restriction is a still more profound restriction of ego activity; in other

words, here the restriction not only applies to the emotions, feelings, desires, in other words the id, but is extended upon the ego itself. This is obviously a defense motivated by the fear that the ego itself may take over some of the dreaded and forbidden activities. This ego restriction manifests itself in a constriction and impoverishment of normal fantasy life with a resulting preponderance of concrete attitudes and thinking patterns over abstract ones. It occurs as a reaction to severe stress, internal as well as external. Among the external stress capable of producing marked ego restriction in otherwise normal personalities is the stress of participation in combat (Alexander and Ax¹).

Projection is the displacement of a forbidden, objectionable, or unacceptable wish, desire, thought, or fact away from the core of the individual's personality either into a more peripheral part of himself, such as a part of his body, or entirely into the outside world. Thus, feelings of inadequacy may be displaced from one's inner self to one's back, one's legs, or one's face. When unacceptable sexual desires or hostile feelings are displaced or projected into the outside world, they may be merely disowned, the individual remaining capable of enjoying their gratification by vicarious identification with a person or persons onto which they were projected, or the projection may go so far as to reverse the aggressor and the target, the patient fancying himself as the passive innocent victim of the very sexual or aggressive desires which he had displaced and projected. Thus projection may range from the widespread habit of enjoying other peoples' love affairs by vicarious identification with the protagonists, to the sinister and pathologic delusion of persecution by one's own projected aggressive and hostile drives, wishes, urges, and desires. Thus projection is a farther reaching and more intricate process than mere displacement. In displacement only the target is exchanged; an acceptable one for an unacceptable one, a permitted one for a forbidden one. In projection the forbidden or unacceptable impulses themselves are disowned, detached, or their direction reversed. The individual's role in the forbidden or unacceptable impulse becomes limited either to a mere vicarious gratification or to passive masochistic fantasy or delusion.

Fearful overalertness is self-explanatory. It indicates an increasing precariousness of the defensive operations, this particular one being a pre-occupation with the readiness for defense indicating eruption into consciousness of the need for defensiveness. It represents an alarm, an alerting oneself to the needs of other defense requirements, a calling for other defensive resources, rather than constituting a defense in itself. It is the last alarm, as it were, before the breaching of the defenses.

Generalized inhibition involves not only the unconscious drives and the ego but also the motor apparatus. It is a rather desperate form of passive defense equivalent to the "Totstell reflex," the playing dead reflex of frightened animals. It implies the individual's giving up or losing confidence in any active defensive stand.

Regression is the abandoning of the mature and highly integrated behavior and reaction patterns of adult life for the primitive reactions of childhood or of primitive and poorly integrated social organizations. This defense is actually a retreat in which valuable equipment and supplies are abandoned to the enemy.

Delusion formation indicates a real and complete breaching of defenses,

since the very wishes, desires, and thoughts which more effective defenses have sought to keep at bay run rampant and erupt into the open territory of the no longer defended ego in the form of wishful gratifications by fantasy. The boundary between reality and fantasy is overrun and reality testing is abandoned. The dream world erupts into the waking life.

Turning against the self may be compared with the outbreak of revolution after the war is lost.

Introjection may be compared with the final conclusion of a contest by Trojan horse tactics, the enemy conniving with traitorous forces within.

Table 4. Defense Reactions

TYPE OF DEFENSE	EFFECTIVENESS OF DEFENSE	PHASE OF DEFENSIVE OPERATION IN RELATION TO STRESS
Sublimation Concentration Identification	Highest level; flexible defensive operations	Adaptation
Displacement Reaction formation Isolation Denial Reversal	Highly effective inflexible defenses	Struggle
Intellectualization Undoing Procrastination Repetition Repression	Moderately effective inflexible defenses	
Inhibition (specific) Anticathexis Ego restriction Projection Fearful overalertness	Borderline ineffective (last-ditch) defenses	
Inhibition (generalized) Regression Delusion formation Turning against self Introjection	Ineffective defenses (implying surrender rather than defense)	Exhaustion

Introjection consists in the unconscious incorporation in the individual's own ego of an ambivalently hated and loved parental figure. This introjected figure is also unconsciously perceived as ambivalently hating and loving, the hateful aspects gaining increasing preponderance as the process of introjection progresses. Thus not only is the perceived hatred of the introjected person allowed to invade the boundaries of the patient's own ego, but also the patient's own hatred of the introjected person is let loose blindly within his ego boundaries, resulting in a battle of annihilation within the inner citadel of the ego, comparable to the destruction of Troy. More about the important and complicated concept of introjection will be said later in this chapter. It is of particular importance in depressions.

The defenses may be viewed from the psychobiologic point of view in

the same light as any other reaction to stress, in that they may represent the three phases of adaptation, struggle, and exhaustion, respectively (Table 4). The first group of defenses may be regarded as reactions in terms of adaptation. They are flexible defenses, of a rather high order. Among these I should like to include: sublimation, concentration, and identification. These actually are part of normal effective living in our human society. Whatever element of struggle there must have been once implied in these defenses seems to have ceased, the stress having been successfully overcome, resulting in adaptation.

The next set of defenses appears to be representative of a phase of continuous struggle, and probably for that reason the defenses in this group are particularly inflexible and rigid. Some of these are quite highly effective, allowing the struggle to be hardly noticeable on the surface, although the psychiatrist who can tell the strain, particularly from the compulsive rigidity of the attitudes they represent, realizes that these defenses put those who are harboring them "out on a limb." The types of defenses in the relatively most effective group of this category are: reversal, reaction formation, isolation, denial, and displacement. Some of these are still almost normal reactions, particularly isolation and displacement, as well as some degrees of reaction formation.

Only moderately effective, although inflexible, defenses in the next sub-category of struggle are: intellectualization, repression, undoing, procrastination, and compulsive repetition. The low borderline of effectiveness among this group of defenses, representing as it were a last ditch struggle, is reached in the following: anticathexis, certain specific inhibitions, ego-restriction, fearful overalertness, and projection.

The phase of exhaustion is represented by defenses which have become entirely ineffective, thus really implying surrender rather than defense. These are: generalized inhibition, regression, delusion formation, turning against the self, and introjection. These are "low" ineffective defenses which actually allow the torrent of destructive and regressive drives full sway. They are actually no defenses at all, unless one could call unconditional surrender a last act of defense after a lost war.

This differentiation of the defensive operations according to their relative effectiveness and according to the phase in the struggle between the emotional needs and the ego which they represent has never been stated with the definiteness which I feel is its due, although a good deal of it is implied in psychoanalytic literature. Anna Freud⁶ has subdivided the defenses into those relating to external danger, such as denial, reversal, inhibition, and fearful overalertness, and those mainly directed against danger from within, such as repression, reaction formation, restriction of the ego, and intellectualization. It may be useful also to make a further division as to whether the defenses are related mainly to affective relationships, to object relationships, or more specifically to certain interpersonal relationships.

The evaluation of the defensive operations is an important step preparatory to and throughout psychotherapy. The nature of the defenses allows important conclusions about the specific orientation of the patient's ego, as well as indirectly and subsequently about his emotional needs. The flexibility or inflexibility, the strength or weakness of the defenses, allows important conclusions as to the patient's ego strength, the phase of the

struggle in which he finds himself, and the type of therapeutic help which he needs. Briefly stated, psychotherapy initially consists in giving the patient's ego sufficient support so that he can temporarily abandon these defenses, a process in which the defenses soon become the core of his resistance to psychotherapy. The object is to render the defenses more flexible or to replace poor inflexible defenses with better and flexible ones. When the defenses are low and ineffective, however, thus showing that the struggle has ended in exhaustion, such as is usually the case in most major psychoses, and particularly in the depressions, the task of psychotherapy becomes well nigh insurmountable.

Depression. Depression probably provides the best example of an illness in which the defenses have been shattered and levelled to the ground. Fenichel⁵ has described the psychodynamic constellation of the depressed patient most lucidly. He holds that pathologic depressive states tend to occur in people with marked oral orientation (whether they have always been that way or whether they have regressed to that stage), whose self-esteem is dependent on the amount of love and esteem they get from others. He calls them "love addicts." In normal development, this stage in which a child's self-esteem is dependent on the love he gets from others is quite an early one, preceding superego development. Normally, the superego takes over the function of regulating self-esteem, offering approval or disapproval according to the now internalized standards of behavior; thus the superego has the dual function of offering protection and approval and of giving a warning signal in the form of guilt feelings when the "narcissistic supplies" are in danger. In these people, however, because of their immaturity or regressed state, the superego is generally rigid and cruel, quite lacking in the ability to protect; their self-approval must come from outer sources and they will do almost anything to get it. This is the main purpose of most of their interpersonal ties, which are further disturbed by the extreme ambivalence arising from their predominantly oral, and secondarily anal orientation. Precipitating factors are generally experiences leading to loss of self-esteem or the loss of or disappointment in a person from whom the patient had hoped to be given the supplies of love and approval he needed. It is the oral fixation that determines the reaction to narcissistic shocks.

Depression occurs when such a person introjects the unsatisfactory person or persons. This introjection or incorporation is a sequel of the marked oral orientation with its incorporating tendencies. It involves a deep regression in which the struggle is removed from the actual person-to-person sphere to become an internalized and hence narcissistic one. The positive aim of the introjection is to get the narcissistic supplies denied in real life. At this stage of regression, however, the superego has become a primitive and cruel force that is incapable of granting approval or forgiveness and which now no longer elicits guilt feelings merely as a signal but instead unleashes the full power of guilt (quite analogous to the panic found in other states), so that in Fenichel's term, the person experiences a sort of "annihilation." This occurs because the existing guilt feelings are augmented by those arising from the introjection of the ambivalently loved person which is perceived as partly sadistic, and by turning against the self of reproaches that apply to the hostile aspects (conscious or unconscious) of the patient's ambivalent relation to the

introjected person, as well as by the reproaches the introjected person might have directed or did direct against the patient.

Fenichel concludes: "Thus the introjection at the basis of depression really is the opposite of the defense mechanism of projection: the bad characteristics of an object which one dare not become aware of because one fears the hatred they would arouse (presumably in oneself) are perceived in one's own ego instead." And "in melancholia it seems as if the main emphasis of the personality has been shifted from the ego to the superego. The patient's conscience represents his total personality; the ego altered by the introjection is the mere object of this conscience and is entirely subdued by it."

Thus, from my point of view, in depression the patient, while not giving up all defenses, resorts to defenses which are so very primitive and ineffective that they are, in fact, nonexistent and the opposite of a real defense mechanism, i.e., they amount to an act of surrender. This in conjunction with the deep narcissistic regression may have a great deal to do with the difficulty in getting through to a depressed patient by interpersonal psychotherapy. *Mutatis mutandis*, the same applies to the other major psychoses, especially to the regression, profound general inhibition, and delusion-formation of the nuclear category of schizophrenia. The fact that some paranoids remain accessible to psychotherapy is probably because projection mechanisms are a better form of defense to work with therapeutically than other forms of delusion-formation. (See Table 4.) The results of psychotherapy indeed parallel the degrees of ego strength and strength of defensive operations in the various diagnostic categories.

Results of Psychotherapy. Adequate statistical evaluations of the effectiveness of psychotherapy are surprisingly rare, and still rarer are studies including an adequate follow-up period. Miles, Barrabee, and Finesinger¹⁷ have recently published an important study of the results of psychotherapy administered in the in-and-out patient department of a general hospital in 62 cases of anxiety neuroses with a follow-up period of from 2 to 12 years. They found 8 per cent apparently recovered; 8.1 per cent markedly improved; 35.5 per cent definitely better and thus considered improved; while 42 per cent were considered essentially unchanged. Only one case (1.6 per cent) included in the latter group was considered worse. Miles and his coworkers also performed a very useful service in tabulating the results of other similar studies done in the past, although most of them are based merely on the status after discharge rather than on actual follow-up. One of the few exceptions is Ross's study²⁰ which included a follow-up period of one year.

Reports of the results of psychoanalytic treatment were slow in coming. Possibly stimulated by the challenging reports on the small groups studied by Kessel and Hyman¹² and Hyman,¹⁰ Knight¹³ published an "Evaluation of the Results of Psychoanalytic Therapy" in 1000 cases. These included the 29 cases previously published by Hyman and Kessel,^{10, 12} as well as the case material from the Berlin Psychoanalytic Institute, the London Clinic, The Chicago Institute of Psychoanalysis, and the Menninger Clinic. The overall rate of improvement in all this case material, the improved total being defined as the sum of the subgroups "apparently cured" and "much improved," was: 63.2 per cent for psychoneuroses, 48.5 per cent

for sexual disorders, 56.6 per cent for character disorders, 78.1 per cent for psychosomatic and related disorders, and 25 per cent for the psychoses.

Table 5. Intensive Psychotherapy of Psychoneurotic and Psychopathic States, Manic-Depressive and Involutional Psychoses, and Others. Complete Recovery and/or Social Recovery, Total Significant Clinical Improvement

TOTAL PTS. ILL	DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
		CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	Total Sig. Clin. Imp.	OF TOT. SIG. CLIN. IMP.
					CR plus SR	Total Sig. Clin. Imp.	CR plus SR
520	Psychoneurosis	314	134	448	60.4	86.3	70.0
92	Psychosomatic	47	14	61	51.2	66.4	77.1
103	Psychopathy	48	33	81	46.6	78.6	59.8
33	Sexual disorder	16	13	29	48.5	87.9	55.2
29	Alcoholic	8	7	15	27.6	51.8	53.3
208	Manic-depressive	97	33	130	46.6	62.5	76.1
47	Involutional melan- cholia	12	10	22	25.5	46.8	54.5
14	Paranoid involu- tional	5	4	9	35.7	64.2	55.5
168	Others	43	26	69	25.6	41.2	62.4
Total 1114		590	274	864	52.9	77.5	68.3

Table 6. Intensive Psychotherapy of Psychoneurotic and Psychopathic States, Manic-Depressive and Involutional Psychoses, and Others. Complete Recovery

TOTAL PTS. ILL	DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE	
		CR	SR plus IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
					CR	CR
483	Psychoneurosis	171	249	420	35.4	40.7
32	Psychosomatic	15	16	31	46.8	48.4
95	Psychopathy	14	63	77	14.7	18.2
33	Sexual disorder	12	17	29	36.3	41.4
19	Alcoholic	3	11	14	15.8	21.4
37	Manic-depressive	6	19	25	16.2	24.0
0	Involutional melancholia					
5	Paranoid involutional	0	3	3		
0	Others					
Total 704		221	378	599	31.4	36.9

The last figure is equal to that reported for spontaneous remission rates in the psychoses.

A number of additional studies are available. Jameison and McNiel¹¹ studied 17 psychotic patients who had either been treated by psychoanalysis for their psychosis or were under psychoanalysis at the time of onset of the psychosis. These authors considered psychotherapy of no value in psychotic states, and in some cases of neurotics under treatment, they felt it may have precipitated the psychosis. Other important studies

Table 7. Intensive Psychotherapy of Schizophrenia. Complete and/or Social Recovery, Total Significant Clinical Improvement

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
						OF TOTAL NO. ILL		OF TOT. SIG. CLIN. IMP.
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	CR plus SR	Total Sig. Clin. Imp.	CR plus SR
	191	Paranoid	26	28	54	13.6	28.2	48.1
	185	Catatonic	55	24	79	29.7	42.7	69.6
	58	Hebephrenic	7	14	21	12.0	36.2	33.3
	30	Simplex	4	6	10	13.3	33.3	40.0
	999	Unclassified	176	219	395	17.6	39.6	44.5
Total	1463		268	291	559	18.3	38.2	47.9

Table 8. Intensive Psychotherapy of Schizophrenia. Complete Recovery

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGES	
						OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	CR	CR
	191	Paranoid	8	46	54	4.2	14.8
	185	Catatonic	19	60	79	10.3	24.1
	58	Hebephrenic	1	20	21	1.7	4.8
	30	Simplex	0	10	10	0	0
	383	Unclassified	62	78	140	16.2	44.2
Total	847		90	214	304	10.6	29.6

are those by Hamilton and Wall⁸ on the effect of psychotherapy on psychoneuroses, by Bond and Braceland² on various psychoses, as well as three studies of the effect of psychotherapy in schizophrenia (Cheney and Drewry⁴, Malamud and Render,¹⁴ and Rennie¹⁸). If these data are taken as a whole, 2577 cases treated with intensive psychotherapy are available for statistical analysis and for comparison with 12,716 controls treated with custodial and supportive care alone, except for the field of psychoneurotic and psychopathic states for which no adequate controls are available. For the purposes of this tabulation, the "character disorders" have been added to the psychopathies (Tables 5 to 12).

Definitions of Grades of Improvement. In defining improvement throughout this book I have—in describing my own results, as well as in cataloguing reports from the literature—distinguished three grades of improvement, as well as therapeutic failure. An unimproved patient

Table 9. Controls—Custodial and Supportive Treatment. Complete and/or Social Recovery, Total Significant Clinical Improvement

TOTAL PTS. ILL	DIAGNOSIS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE		
		CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.	
116	Manic-depressive; de- pressed	77	6	83	66.4	71.6	92.8
0	Manic-depressive; manic						
112	Involutional melan- cholia	16	32	48	14.3	42.8	33.3
113	Involutional psychosis (paranoid)	49	13	62	36.9	46.6	79.0
0	Other psychoses						
0	Psychoneurosis						
Total	341	142	51	193	41.5	56.5	73.5

Table 10. Controls—Custodial and Supportive Treatment. Complete Recovery

TOTAL PTS. ILL	DIAGNOSIS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE	
		CR	SR plus IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
88	Manic-depressive; de- pressed	15	32	47	17.0	31.9
0	Manic-depressive; manic					
93	Involutional psychosis	43	0	43	46.2	100.0
0	Other psychoses					
0	Psychoneurosis					
Total	181	58	32	90	32.2	50.1

(therapeutic failure) is one who is either still in the hospital or a home invalid, or one who has been in any one of the other categories before treatment but who has failed to advance with treatment to a higher grade of improvement. An improved patient is one who has sufficiently improved to be discharged from the hospital or who is no longer a home invalid; who is capable of meeting some, but not all, requirements of his social and occupational life. A social remission or social recovery de-

scribes a patient who has sufficiently improved to resume his social and occupational activities to the full, but who has either failed to achieve insight or still retains some subjective complaints or disturbances in his intimate personal life. Complete recovery denotes the restoration of the mental state of the patient to what he and his relatives agree is his former

Table 11. Control Patients—Custodial and Supportive Treatment of Schizophrenia. Complete and/or Social Recovery, Total Significant Clinical Improvement

Breakdown by Diagnosis								
TOTAL PTS. ILL		DIAGNOSIS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE		
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.	
	1035	Paranoid	124	115	239	11.9	23.0	51.8
	801	Catatonic	244	60	304	30.4	37.9	80.2
	566	Hebephrenic	86	67	153	15.1	27.0	56.2
	54	Simplex	13	8	21	24.0	38.8	61.9
	8624	Unclassified	1636	919	2555	18.9	29.6	64.0
Total	11,080		2103	1169	3272	18.9	29.3	64.2

Breakdown by Duration of Illness*								
TOTAL PTS. ILL		DURATION OF ILLNESS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE		
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.	
	1445	1-18 months	678	124	702	47.0	48.5	96.7
	834	Over 18 months	152	79	231	18.2	27.7	65.8

* Patients reported in this table are a duplication of a portion of those patients reported above under "Breakdown by Diagnosis."

"own best self," implying that such full recovery is associated with insight as well, although insight may not be entirely lacking in other degrees of improvement.

Results of Psychotherapy in the Major Psychoses. If we compare the figures for psychoses for which controls are available, we find no significant difference for all three categories of improvement (complete recovery, complete and/or social remission, total significant clinical improvement) in the cases of manic-depressive psychosis (compare Tables 5 and 6 with Tables 9 and 10), between the cases treated with custodial and supportive care, and those treated with intensive psychotherapy. Particularly the rate for complete recovery is practically identical (16.2 vs. 17 per cent).

Neither is there any significant difference in the rate for complete and/or social remission between the group of schizophrenic patients treated with intensive psychotherapy and those with custodial and supportive treatment respectively, the rates being 18.9 and 18.3 respectively (compare Table 7 with Table 11).

Table 12. Control Patients—Custodial and Supportive Treatment of Schizophrenia
Complete Recovery

(Figures appearing in the two tables below do not represent the same patients)

Breakdown by Diagnosis							
TOTAL PTS. ILL.		DIAGNOSIS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE	
						OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	CR	CR
	36	Unclassified	19	10	29	52.7	80.5
	449	Paranoid	3	84	87	0.7	3.5
	355	Catatonic	28	69	97	7.9	28.9
	206	Hebephrenic	2	30	32	1.0	6.3
	24	Simplex	2	9	11	8.3	18.2
Total	1070		54	202	256	5.0	21.9

Breakdown by Duration							
TOTAL PTS. ILL		DURATION OF ILLNESS	BREAKDOWN OF CLINICALLY IMPROVED PATIENTS			PERCENTAGE	
						OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	CR	CR
	22	1-18 mos.	3	17	20	13.7	15.0
	14	18 mos.-4 yrs.	0	8	8	0.0	0.0
	24	Over 4 yrs.	0	6	6	0.0	0.0
	165	Duration not stated	8	58	66	4.9	12.1
Total	225		11	89	100	4.9	11.0
Grand Total 1295			65	291	356	5.0	18.2

However, the rate for full recovery is significantly higher in the group treated with intensive psychotherapy: It is 10 per cent for that group (Table 8), as compared to 5 per cent in the group treated with custodial and supportive treatment only (Table 12). This difference is significant below the 0.01 level, i. e., there is less than one chance in one hundred that this difference is due to chance sampling alone. We are forced to conclude from this finding that the addition of intensive psychotherapy

to the therapeutic regimen in schizophrenia does not increase the number of patients who are going to improve, but helps a significant portion of those patients who are improving to achieve a better quality of improvement.

This finding illuminates the value of psychotherapy as an ancillary technique in the treatment of the major psychoses in that it enhances the quality rather than the rate of improvement, and gives us adequate reason to resort to physical treatment for the relief of the major psychoses themselves, but to psychotherapy for bettering the quality of the remission thus initiated. An exception would be certain cases of schizophrenics who actively struggle with the neurotic conflict and thus remain reasonably accessible. Psychotherapy also remains an important technique in helping the patient improve his defensive position after recovery from the psychosis, irrespective of whether this came about spontaneously or was brought about by physical treatment.

PSYCHOPHYSIOLOGIC EFFECTS OF PHYSICAL TREATMENT

Psychophysiologic Effects of Electric Treatment

Electric therapy is now widely used in the treatment of depressions and other psychotic states, but the most prevalent method—convulsive electroshock—has certain disadvantages. These disadvantages are that while convulsive electroshock relieves depression, it frequently enhances anxiety and reduces awareness for memory contents. A more recent development of treatment—nonconvulsive electric stimulation—can be used alone or in conjunction with convulsive therapy to overcome these disadvantages.²⁸

Convulsive Treatment. *The Effect of Convulsive Electroshock on a "Normal" Person Recently Subjected to Stress.* Before discussing the specific actions of electric convulsive therapy in mental diseases, it may be well to consider its effect on "normal" persons, that is, on persons not complaining of or significantly disturbed by mental and emotional conflicts (being fully aware, of course, that a supremely placid and non-disturbed individual does not exist). For obvious reasons such observations are rare. On one occasion, however, chance played into my hands so that I was able to make such an observation.²⁹

A man, 50 years old, of distinctive accomplishments in a field closely related to my own, and who was therefore frequently in close contact with patients who are about to undergo or have recently undergone an experience of electric shock therapy, consulted the superintendent of the hospital, who referred him to me, with a request that he be allowed to undergo electric shock therapy, in the hope that this would render him more capable of understanding the frequent qualms and resentments of his clients. He hoped that a personal experience of undergoing an electric shock would enable him to know better whereof he spoke with his clients. The request was unusual but rather well rationalized. There was no overt mental or emotional disturbance in this man, although an undertone of desire to undergo punishment may have been implied by the fact that he and his organization had recently undertaken an ambitious venture that ended in conspicuous failure. This circumstance had persuaded him to resign from his position and to accept a similar position elsewhere. However, he did not mention the failure and I tactfully refrained from doing so likewise. He explained that he had brought his work in the town where he had been working to conclusion and that he was eagerly looking forward to starting similar work in some other locality in which development in our field was

just about ready to surge forward. I hesitated to give the treatment for more than one reason. I put him through an elaborate series of physical fitness tests and in general tried to put him off, at least until I might discover some way in which I could justify the granting of his request by setting up a planned experiment that would contribute scientific data commensurate to the strain both on the subject and on myself. In the meantime he pressed me to go ahead with the experiment, for the time of his departure moved closer. On the day set for the treatment, chance played into my hands in providing an ideal experimental test situation. On his frequent visits to my research clinic, this gentleman had usually driven in himself in his own car. This particular morning he arrived without the car, having lent it to his daughter. He was in a state of considerable upset, for he had just been through a rather disturbing traffic accident. The street car in which he was riding had hit an automobile, and a particularly harrowing feature was that the street car had hit the motor car twice. The first impact spun the car around, and then the street car, its brakes obviously not working, had hit the car a second time from the other side, throwing the car this time against a telephone pole and jamming it against it. Even the street car was derailed by the impact and the passengers, including our subject, had to continue the journey by a special bus dispatched to the scene.

The subject had arrived some time ahead of me that morning, and he told his story to my secretary who with excellent presence of mind took down verbatim what he had told her about it and showed me the transcript before I saw the gentleman. I realized that this was a perfect experimental situation, and decided to proceed with the treatment then and there. In order not to offer the slightest suggestion, I completely refrained from discussing his recent experience with the patient prior to the treatment, but rather breezily took him off to the treatment room without allowing him to discuss the accident with me at all.

A combined convulsive-nonconvulsive treatment of a total of three minutes' duration was administered through temporoparietal leads. The treatment went off smoothly and without incident. He was cheerful and well at ease upon awakening, stating that he was fine except for the calves of his legs which felt as though he had a charleyhorse. He was rather proud of remembering numerous details preceding the treatment. He remembered that the nurse put jelly on his temples and also that the doctor readjusted the position of the head band, taking it off once in order to fit the electrodes more perfectly into place. He also remembered the name of the attendant who stood on his right and that of the nurse who stood on his left side. He remembered what he had for breakfast that morning and that he had come by bus rather than in his own car. I asked him whether anything unusual had happened on the way and he said, "Nothing at all." "Was there an accident?" I asked; "none that I know of," was his reply. I asked him whether he had a conversation with my secretary and he said he remembered talking with her a little and giving her his newspaper, which was his usual custom on visits to the clinic. He added, "My mind feels as clear as a bell."

The following ten days the subject felt fine, going about his usual activities and making all the arrangements for his departure. He noted more or less incidentally that he and his family felt that he was more relaxed and more jovial than he had been for some time. Ten days after the treatment I asked him again about the events on the treatment day. He again emphasized that he remembered everything about the treatment: lying down on the bed, the jelly on his temples, and the adjustment of the strap, the attendant holding one shoulder, the nurse holding the other. He could remember details after the treatment: waking up, smoking a cigarette (he also remembered that the cigarette had no taste to it, an observation frequently made by patients who have recently experienced an electric shock), that he was lying on the bed in the recovery room feeling well at ease, and knowing precisely where he was. I

then asked him again about the events preceding his arrival that morning. He remembered getting up at 8:30, then having breakfast. This time he stated that he then drove to the hospital in his own car, arriving at 9:30; that he read his newspaper for a while before the doctor arrived and took him to the treatment room. I then asked if anything unusual had happened on the way to the hospital, and again he replied that he could recall nothing unusual. I asked him, "Do you remember the topic of your conversation with my secretary before I arrived?" He answered, "I don't remember what the conversation was about." "But what about the accident?" I asked. Surprised, he replied, "I had an accident? I don't recall that at all."

The verbatim account of his story as he had given it to my secretary on the morning of his treatment was then read to him. He smiled and then said, "Oh well, I recall that accident very clearly. Of course, I remember exactly that I had taken the street car that morning because my daughter needed the car. It is all clear in my mind now, and was clear in my mind all along, except that I didn't think of it, and if I may have thought of it—which I don't remember now—I must have thought of it as something that happened sometime ago, without connecting it with that particular morning or with my present life. But, of course, I remember every detail of it." The patient then redescribed the accident in a calm manner with all the pertinent details, emphasizing that he had full recollection of the accident but that it no longer had any emotional meaning for him.

This observation brings up important points of interest. First of all, the accident as well as the street car ride were the only items for which there was amnesia immediately after the electric treatment. Earlier as well as later events were clearly remembered then. Ten days later, when confronted with the account he had given the secretary before his treatment, he was able to call forth a very clear memory image of the accident. It became obvious that all that had actually been lost was the associative connection of the accident with the present time and his present emotional life. Thus this selective amnesia is clearly not an amnesia in the ordinary sense at all, but the "symbolic amnesia" inherent in the defensive operation of isolation which Freud⁶⁹ had defined as the "stripping" of the associative connections from a traumatic or disturbing event or mental content that once linked it with the remainder of the active mental life, thus depriving the event or mental content of its affective meaning. It becomes clear therefore that what has happened in this case is not simple forgetting, but rather the result of the bringing into play of the defensive force of isolation.

The Psychophysiologic Effect of Electroshock on Abnormal Mental States. In abnormal mental states, it is the arousal or enhancing of active defensive operations that seems to be the most decisive effect of convulsive electroshock treatment. It is probably for that reason that convulsive electroshock therapy is helpful in those conditions in which defensive operations are at a low ebb, ineffective, or exhausted (see p. 197), as in depressions and some other severely regressive psychotic states, but that it is ineffective—in fact disturbing—in those conditions in which defensive operations are already too disturbingly alerted, constituting an obstacle to treatment and recovery, such as in anxiety states.

The Fundamental Differences between Anxiety and Depression. In this connection I must explain my mutually exclusive use of the terms depression and anxiety. To be sure, anxious thoughts occur in depression,

and depressive thoughts in anxiety, but when we look beyond the superficial verbalizations of content the two disturbances are fundamentally different from each other. First of all, anxiety is a state of excitation, depression a state of inhibition. The anxious patient feels and acts like a prisoner about to escape, or a fugitive fleeing for his life, running and hiding with bated breath and beating heart, thus by his actions betraying that in spite of his fear, he has remained essentially optimistic about escaping his doom. The depressed patient is like a prisoner who has accepted his doom. He knows or assumes that there is no escape; unconsciously he even longs for the end. He fears it, to be sure; but his muscles do not quiver, his heart does not beat fast, his throat does not choke; he feels empty and dead inside.

In more scientific terms, Freud⁶⁹ has formulated anxiety as the reaction of the mental apparatus to excessive quanta of excitation, in addition to being a signal of danger, or a reaction to the danger of a loss of or a separation from a cherished object. Freud emphasizes that motor innervations, efferent processes, as well as physical sensations from the inner organs, such as the respiratory organs and the heart, take part in the total phenomenon of anxiety. He enumerates the chief attributes of anxiety as: (1) a specific unpleasurable quality, (2) efferent or discharge phenomena, and (3) the perception of these. He continues (p. 70): "Anxiety, therefore, is a specific state of unpleasure accompanied by motor discharge along definite pathways. In accordance with our general outlook, we shall believe that an increase in excitation underlies anxiety, an increase which on the one hand is responsible for its unpleasurable character and on the other is relieved through the discharge referred to." He adds that anxiety is anticipation of trauma, as long as there is still time to avert it; "a call for help in the danger situation," the ego taking into its hands the direction of the course of the trauma. In depression—mourning, grief, and melancholia—on the other hand, the ego is paralyzed and defenseless. "Grief is therefore the reaction specific to object loss, anxiety to the danger which this object loss entails, or, by a further displacement, to the danger of object loss itself," (p. 119). In other words, grief and depression are reactions to the accepted fait accompli, anxiety merely to the danger which is not accepted and which the individual strives to forestall.

Recent neurophysiologic research has further confirmed the fundamental difference between anxiety and depression (Funkenstein et al.⁷¹⁻⁷⁴). Anxiety can be precipitated or increased by the injection of epinephrine, while depression cannot. Neither can the anxiously colored feelings and thoughts of the true depression be enhanced by epinephrine; when there is a true admixture of anxiety, however, such as exists in certain atypical depressions and in schizo-affective states, this true anxiety can be increased or precipitated by epinephrine. A recent case of my clinical observation supplies a striking example. A 57 year old, hitherto vigorous, energetic, and healthy man was referred to me as an anxiety state, because his complaints centered about his heart and other body organs. He knew that he was through; that his days of health and manhood had passed. He assumed that his heart would not last long, or that his high blood pressure (which had been found moderately elevated shortly before the onset of his illness) would burst a blood vessel of his brain. He only hoped that this would be the end, so that he would not have to live on as a para-

lyzed invalid. All these thoughts were uttered slowly, sadly, the patient sitting down motionless with a stony facial expression. He obviously had accepted his doom. His pulse rate was not elevated, although his blood pressure was. The total clinical picture was certainly that of a depression, in spite of the superficially anxious content. This clinical impression was confirmed by the fact that an injection of epinephrine precipitated no true anxiety, and no increase in his depressive pseudo-anxious thoughts and feelings. Convulsive electroshock treatment brought about prompt and complete recovery, without build-up of anxiety, which would have been inevitable had there been any true anxious agitation connected with his illness. In this book I shall therefore limit the use of the words anxiety and depression respectively to the specific phenomena defined above, both in their psychiatric and physiologic-pharmacologic aspects.

The Clinical Effects of Convulsive Electroshock in Mental Diseases. Convulsive electroshock relieves depression in an almost specific manner, and with remarkable promptness. It never ceases to be a source of astonishment and delight to see a patient who had been severely depressed for months, tormented by self-hate and self-recriminations, feeling cast out and abandoned by a world perceived as odious and punitive, as a vale of tears and misery, envisioning himself as burning in hell with the damned, after one to six electric treatments reaccepting himself and the world as good and the Deity as loving and generous.

Recently an outstanding businessman, nationally renowned for great enterprise, fairness, and high ethical standards which had gained him wide recognition as a sought-after expert in his field, had developed a crushing belief that he was nothing but a worthless cheat, and could actually see and feel himself burn in hell, a visual eidetic experience which was probably facilitated by the fact that as a child he had actually seen his mother vigorous and healthy man's depression was of six months' duration and for four months had been of maximally severe degree, replete with the most agonizing self-torment and self-debasement, before he came to see me. After one convulsive electroshock treatment, this man's natural self-acceptance was restored. When the patient was confronted with the ideas held by him only the day before, he said with a smile and considerable amused animation, "Well, I don't worry about my sins any more—I know I have plenty of them; it is no joke, but I imagine a lot of people have more than I." Such dramatic recoveries of severe and otherwise resistant mental illnesses entailing deep suffering can be brought about with practically no other treatment in psychiatry. This effect on depression is involuntary, or reactive. Electroshock is also very effective—to an almost equal or slightly less degree—in other conditions in which defenses are either insufficient or inoperative. This is particularly true of acute catatonic states as well as other schizo-affective states in which schizophrenic and affective features commingle. Electroshock is also highly effective in manic conditions, although less readily than in depressive states, more treatment being required.

Electroshock is also helpful in other unspecific acute psychoses with delusional and hallucinatory features, including organic psychoses such as those induced by involutional or senile disturbances, neurosyphilis,

drugs, as well as in certain psychoses at the borderline of the organic which are akin to epileptic twilight states, which Kleist¹⁰² had described as "episodic twilight states," and which Meduna and McCulloch¹¹¹ recently renamed "oneirophrenia." These psychoses—which are rare—are characterized by elements of definite clouding of consciousness which may easily escape detection amidst the general picture of delusional disorientation. I am sure a good many of them have crept into the diagnostic categories of acute catatonia or the various other schizophrenic psychoses. They are probably not numerous enough, however, to affect the figures decisively. I see about one or two every few years. While their average spontaneous duration may last from one to six weeks, electroshock certainly curtails the duration to as little as a day or two. They generally clear up with one or two treatments. Undoubtedly, some of the cases requiring very little electroshock treatment fall into that category.

I have found electroshock to be effective also in various paranoid states, particularly in the paranoid involutional group.

All those conditions in which electroshock is effective have in common the fact that defensive operations are at a low ebb. Improvement is associated with improvement in the quality of the defensive operations. It is of interest that while defenses become re-erected following electroshock, they also appear to become more flexible, thus facilitating psychotherapy and a general widening of the patient's adaptive potentialities. Electroshock is least effective in neurotic states and in the general group of schizophrenic states in which it is inferior to other forms of treatment, namely to psychotherapy in most cases of neurosis, and to insulin coma treatment in most cases of schizophrenia. This is probably due to the fact that in these conditions the defensive operations are alerted, frequently overactive, thus making the increment from convulsive electroshock disturbing rather than helpful.

We must, however, not be dogmatic in the use of diagnostic labels. Psychodynamic evaluation of the defensive position of the patient as well as the evaluation of the pattern of responsiveness of the autonomic nervous system will be the best lead in deciding whether electroshock will or will not be helpful in the particular phase of the patient's illness.

Statistics of Treatment Results. The overall statistical results of convulsive shock therapy are impressive. Impressive also is the great number of patients concerning whom statistical data of treatment results are available in the literature. Since Ziskind's¹⁵⁸ last survey of the literature in 1946, reporting on a total of 2777 cases, 11,304 more have become available, both by adding a number of reports antedating Ziskind's study, as well as adding more recent ones, thus making a total of 14,081 cases* of convulsive shock therapy. Of these 9312 are definitely specified as having been electroshock, and 4146 are specified as metrazol, cardiazol or pentrozol, while in the remaining cases, authors do not definitely state whether the convulsions were induced by electricity or by metrazol or its derivatives. The group for which control observations of cases of the same diseases observed by the same authors but not treated with electroshock or

* See refs. 32, 33, 34, 35, 37, 38, 45, 46, 48, 49, 50, 51, 53, 54, 57, 58, 59, 60, 63, 64, 67, 76, 78, 79, 80, 81, 82, 84, 85, 86, 87, 90, 92, 94, 96, 97, 99, 100, 101, 105, 107, 108, 110, 116, 117, 118, 119, 120, 123, 125, 126, 127, 128, 130, 131, 133, 135, 139, 140, 141, 142, 144, 145, 148, 149, 150, 151, 153, 155, 156, 157, 158.

metrazol are available is considerably smaller, comprising, however, a total of 1805 cases as compared with 1523 controls.† If all controls, including those described by other authors are included, they amount to 11,421 cases.

The overall significant improvement rate for *depressions of manic-depressive type* (Table 13) is 84 per cent for convulsion-treated patients, (76.7 per cent for the least favorable sub-group) while the parallel control group of non-shock-treated patients achieved spontaneous improvement in 71.6 per cent of the cases (Table 9). Full recoveries (Table 14) occurred in 44.7 per cent of the treated cases, but in only 17 per cent of the control group (Table 10). The differences in this group are even more impressive if only the recoveries which occurred within the first year are considered. Here the recovery rate is only 13 per cent for the control group (Tillotson and Sulzbach¹⁴⁵). The greatest gain in the manic-depressive group attributable to convulsive therapy is thus to be considered the abbreviation of the duration of the illness, which electroshock treatment diminishes by 75 per cent to 90 per cent (Tillotson and Sulzbach,¹⁴⁵ Oltman and Friedman¹¹⁹). It is important to realize, however, that manic-depressive psychosis is by no means entirely recoverable in all cases; spontaneous improvement failed to occur in almost 30 per cent of the control group without shock therapy, while failures were reduced as low as half that rate—namely, 16 per cent—in large groups given electric shock therapy. It is important also to note that many patients who had failed to improve without shock and had become chronically ill have nevertheless responded when subjected to shock treatment many years after the onset of their illness. Tillotson and Sulzbach¹⁴⁵ mention 4 patients, hospitalized with unrelenting depressions for five to fifteen and a half years, who recovered fully in three weeks to four months after the first convulsive electroshock treatment and had maintained their recovery at optimal level for at least two years and three months at the time of the writing of their paper. Other authors also emphasized this point, among them Prosser,¹²⁵ who has noted 3 patients with affective psychoses ill over seven years and one ill over twelve years who gained remissions with electric shock. Nor is recovery with shock treatment dependent on the illness being a first attack (Rennie¹²⁶).

Manic patients show an overall improvement rate of 74.6 per cent after convulsive shock treatment (Table 13), some individual groups showing as low a rate as 30 to 67 per cent, while full recovery occurred in only 46.3 per cent of the shock-treated cases (Table 14). Since no control figures are available for this group, we are unfortunately unable to compare these figures with the rate of spontaneous recovery. Schiele and Schneider,¹³⁷ however, feel that convulsive electroshock is definitely helpful when used early in the course of the manic attack. The lesser degree of responsiveness of manias is probably due to the fact that in certain manic states—especially in irritable mania—there is a marked admixture of anxiety which may become enhanced under convulsive electric therapy. The failure of some manics to respond at all to electroshock is probably explained by this circumstance, as well as by the fact that manic behavior itself is a highly alerted, in fact, frantic, defense. Since defenses in general are alerted rather than levelled off or brought down by the usual thera-

† See refs. 53, 58, 87, 90, 92, 119, 144, 145, 158.

peutic doses of electroshock such as are helpful in depressions, mania tends to be enhanced, or if already maximal, tends to remain uninfluenced by such treatment, unless electroshock is administered in doses large enough—several treatments daily—to produce organoid personality disorganization, thus breaking down with it the defensive operations of the personality. This represents a rather unphysiologic use of electroshock which Fabing^{61, 62} has succeeded in improving on by rendering single electroshocks supramaximal in physiologic effect by preceding the treatment with an intravenous injection of coramine. The opposite approach was introduced by Delmas-Marsalet (p. 72) who used nonconvulsive treatment. I have found this warranted on the basis of the autonomic response pattern (the presence of epinephrine-precipitable anxiety) in certain patients, as well as effective under such circumstances. More about this will be said in the section on nonconvulsive electric therapy.

The fact that mania responds less well to convulsive shock treatment than does depression is interestingly confirmed by Simon and Holt's¹³⁹ study of various specific psychiatric symptoms, and the degree of recovery from these manifestations made by a representative group of psychotics treated with shock therapy:

	%		%
Suicidal attempts	95	Delusions	57
Refusal of food	93	Bizarre speech	57
Untidiness	82	Confusion & bewilderment	55
Bizarre acts	75	Underactivity	51
Refusal to speak	70	Disorientation	50
Depression	69	Lack of interest	50
Anxiety	68	Overt sexual activity	47
Overactivity	66	Inappropriate mood	42
Hallucinations	62	Euphoria	15
Overtalkativeness	60		

Euphoria, a highly effective defense—in fact, one of the strongest defenses—, proved to be the least responsive. Although anxiety rates rather high in recovery according to this survey, this probably refers to anxious thoughts rather than anxiety in the psychobiologic sense of the word as I am using it (see above).

Of patients suffering from *involutional melancholia* 82.2 per cent attained significant improvement with electroshock treatment (Table 13), while only 42.8 per cent improved without such treatment in the parallel control group (Table 9). Paranoid involutional psychoses are slightly less responsive to electroshock than involutional depressives, namely 76.3 per cent for the treated and 46.6 per cent for the untreated group (Tables 13 and 9). As for full recovery in involutional psychosis, Tillotson and Sulzbach¹⁴⁵ report 52 per cent with electroshock and none in the control group. Our combined figures show 53.8 per cent for involutional melancholia, 32.5 per cent for involutional paranoia in the shock-treated group, an average of 46.2 per cent in the control group. Tait and Burns¹⁴⁴ obtained a 60.8 per cent full and social remission rate with 171 shock-treated involutional melancholia patients, while only 8 patients of 101 in the control group gained spontaneous remissions. They found this same disparate ratio to hold true in other involutional psychoses as well.

In *schizophrenia* the improvement rate is much less: 49.1 per cent of the treated group (Table 15) attained this result as compared to 29.3 per

cent in the control group (Table 11). A breakdown of the figures available shows that 48.8 per cent of 598 catatonics attained significant improvement, as did 46.8 per cent of 449 paranoids, but only 31.5 per cent of 346 hebephrenics (Table 15). The figures for full recovery with convulsive

Table 13. Convulsive Shock Therapy. Complete and/or Social Recovery, and Total Significant Improvement

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL		OF TOT. SIG. CLIN. IMP.
						CR plus SR	Total Sig. Clin. Imp.	CR plus SR
	2,165	Man.-dep.; dep.	1471	345	1816	67.9	83.9	81.1
	393	Man.-dep.; man.	240	53	293	61.2	74.6	81.9
	1,136	Invol. mel.	749	185	934	65.9	82.2	80.2
	788	Invol. psy. (par.)	521	81	602	66.2	76.3	86.7
	7,357	Schizophrenia	2135	1469	3604	29.1	49.1	59.2
	1,912	Other psychoses	946	415	1361	49.5	71.3	69.5
	293	Psychoneuroses	123	57	180	41.9	61.4	68.3
	37	Man.-dep.; mixed	29	0	29	78.4	78.4	100.0
Total	14,081		6214	2605	8819	44.2	62.6	70.5

Table 14. Convulsive Shock Therapy. Complete Recovery

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGES	
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
						CR	CR
	732	Man.-dep.; dep.	327	312	639	44.7	51.3
	26	Man.-dep.; man.	12	8	20	46.3	60.0
	119	Invol. mel.	64	40	104	53.8	61.5
	209	Invol. psy. (par.)	68	111	179	32.5	37.9
	2189	Schizophrenia	373	981	1354	16.0	27.5
	115	Other psychoses	29	59	88	25.2	33.0
	59	Psychoneuroses	26	25	51	44.1	50.9
Total	3449		899	1536	2435	26.1	36.9

shock treatment (Table 16) are much lower, 16 per cent, although significantly higher than in the control group, 5 per cent (Table 12). Nevertheless, Myerson¹¹⁷ concluded that electroshock is of comparatively little value in the full-blown schizophrenic state, regardless of diagnostic subcategory, and stressed the fact that the question of differential diagnosis—particularly in patients with early schizophrenia who are the ones who respond best—may have much to do with the interpretation of results.

Rennie¹²⁶ also feels that illnesses of a short duration provide the best prognosis, while Schiele and Schneider¹³⁷ and Gold and Chiarello⁷⁹ have pointed out that, in general, the better the prepsychotic personality, the better the prognosis. Woolley et al.,¹⁵¹ however, claim 45 per cent recovery

Table 15. Convulsive Shock Therapy of Schizophrenia. Complete and/or Social Recovery, Total Significant Clinical Improvement

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
						OF TOTAL NO. ILL		OF TOT. SIG. CLIN. IMP.
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	CR plus SR	Total Sig. Clin. Imp.	CR plus SR
	449	Paranoid	128	82	210	28.5	46.8	60.9
	598	Catatonic	215	77	292	35.9	48.8	73.6
	346	Hebephrenic	64	45	109	18.5	31.5	58.8
	151	Simplex	34	28	62	22.5	41.0	54.8
	5813	Unclassified	1694	1237	2931	29.1	50.4	57.8
Total	7357		2135	1469	3604	29.1	49.1	59.2

Table 16. Convulsive Shock Therapy of Schizophrenia. Complete Recovery

TOTAL PTS. ILL		DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGES	
						OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	CR	CR
	163	Paranoid	23	56	79	14.1	29.2
	206	Catatonic	56	68	124	27.1	45.2
	146	Hebephrenic	8	42	50	5.5	16.0
	52	Simplex	3	23	26	5.8	11.6
	1622	Unclassified	283	792	1075	17.4	26.4
Total	2189		373	981	1354	16.0	27.5

and parole of a total group of 31 chronically regressed patients, and Kalinowsky et al.⁹⁶ report that 3 chronic regressed patients ill over ten years attained and held parole status following electroshock, and 14 more of a group of 56 chronic patients improved and held that improvement.

Among other psychoses, unclassified atypical depressions and schizoaffective reactions, together with some atypical psychoses in the involutional and senile age group as well as symptomatic psychoses, figure most prominently. Here the improvement rate in the treated group is seen to be 71.3 per cent (Table 13), the full recovery rate (Table 14) 25.2 per cent (no controls available).

It is difficult to evaluate the figures on *neuroses*—61.4 per cent in the

treated group (Table 13). This is about equal to the improvement rate attainable with psychotherapy (63.2 per cent, Knight¹³). Unfortunately, untreated control figures by the same authors are not available for this classification. Because defensive operations are notoriously alerted—including such highly effective defenses as isolation and denial—electroshock results are least good in compulsive and obsessive states, which comprise a considerable proportion of the psychoneuroses under consideration here (Chodoff et al.⁴⁶).

It is also quite likely that mild depressive reactions and schizo-affective states may have crept into the psychoneurosis category. Myerson^{114, 115} pointed out the diagnostic difficulties which these mild, technically psychotic reactions offer in their differentiation from neuroses. He called them "neuropsychoses"—psychotic episodes with purely neurotic symptomatology. The importance of this concept was recently confirmed by Bowman and Rose,⁴⁰ who suggest the abolition of the terms "psychosis," "psychoneurosis," and "neurosis" entirely from the psychiatric vocabulary, pointing out that the present-day usage of these terms serves only to distinguish the degree of severity of a mental disorder, a distinction more fairly served in their opinion by the phrases "mild, moderate, or severe reactions." A recent editorial in the *American Journal of Psychiatry*⁴¹ also stresses the diagnostic inadequacy of these terms, pointing out that many a mild depression, clinically a psychosis, may be classified as a neurosis because of its unpronounced symptomatology, a point further emphasized by Campbell⁴⁴ who suggests that many manic-depressive attacks go disguised as seeming psychoneuroses because of their lack of severity. Stone and Burris¹⁴³ reported particularly high recovery rates with electroshock treatment in this group of cases (96 per cent) in spite of long duration of illness (up to 33 months).

A number of 3 to 10 year follow-up studies are available at the present writing. Most authors agree that relapses in successfully shock-treated cases occur neither more nor less frequently than after comparable states of spontaneous recovery.^{64, 65, 82, 87, 92, 113, 130} It is in the follow-up work, however, that psychotherapy comes in most importantly, for when psychotherapy is ineffective in significantly influencing the course of the psychotic episode itself, it is probably effective in preventing relapses. This is an area in which further study is needed. Clinically I am sure it is so; my own experiences in this particular area will be discussed in Chapter XV.

Electroshock and Intractable Pain. Electroshock relieves the somatization reactions associated with some depressive illnesses, especially the displacements and projections in the form of pain ("psychalgia"). These cases frequently go long unrecognized under the guise of apparent physical disease, because the successful defense in the form of displacement and projection prevents the overt eruption of depression for a long time. For the same reason treatment of these cases by electroshock is frequently troublesome, because the further alerting of defensive operations by convulsive electroshock leads to a marked, temporary, sometimes prolonged administration of nonconvulsive treatment, as well as by psychotherapy. More about this will be said later. Electroshock does not relieve pain caused by physical illnesses. It may therefore sometimes be a helpful differential

diagnostic measure in bringing to light a coexistent but formerly overlooked organic disease (Yaskin¹⁵⁴).

Electroshock, however, is reported to relieve the somatopsychic reactions of some systemic illnesses, such as the irritability and severe anorexia of acrodynia (Bernheim and Girard³⁶).

Electroshock Treatment as a Lifesaving Measure. No study of electroshock treatment results would be complete without mention of the dramatic lifesaving quality of shock treatment. Huston and Locher⁹² report that of a control group of 93 patients suffering from involutional psychoses, 12 patients succeeded in taking their own lives, and 21 others died; 9 of this number succumbed to illnesses directly attributable to their psychoses, namely, agitation to the point of exhaustion. One patient in the shock-treated group committed suicide; there were no other deaths. Similarly, Ziskind¹⁵⁸ has recorded 9 suicides in a control group of 109 affective psychoses receiving forms of psychotherapy other than shock treatment, and 4 further deaths in this group from exhaustion, while there was but one suicide in the shock-treated group, and no other deaths. We might note in this connection that suicidal attempts head the list of psychiatric symptoms quoted on page 213 in the order of their rapidity of response to electroshock. In cases of mania in which a high degree of excitement may lead to death from exhaustion, Schiele¹³⁷ confirms the use of electroshock as a lifesaving measure.

Decrease in Hospitalization. A further tangible advantage of electroshock treatment is that it materially shortens average hospitalization time in most psychoses, thus increasing hospital capacity. In one instance (Eitinger⁵⁸) this amounted to an increase in availability of hospital beds by 100 per cent per year. The same author reports a decrease in hospitalization from an average of 839 days among controls to 273 days among the treated group; Huston and Locher⁹² report a reduction from 21 months for control patients to 6 months in the shock group, those of the shock group who responded to treatment averaging four to five weeks. Tillotson and Sulzbach¹⁴⁵ reported similar figures; other authors, including Rennie,¹²⁶ Oltman and Friedman,¹¹⁹ Hinko and Lipschutz,⁸⁷ and Wilson,¹⁴⁹ concurred.

Quality of Improvement. Another point of importance in assaying the value of electroshock, particularly in depressions, is the quality of improvement obtained. It has been my clinical impression that the quality of improvement obtainable with electroshock treatment on the one hand, compared with that obtained by nonphysical methods on the other, especially in depressions from the 40-year age group onward, is better in the group which received electroshock treatment. A much larger number of patients with such midlife depressions than is generally appreciated fail with nonphysical treatment methods to regain their old zest for life, but remain somewhat subdued, somewhat less vital, somewhat less capable of enjoyment. This is difficult to ascertain, of course, in individual cases, unless one either has known the patient intimately from the period before the onset of his depression, or carries out very complete personality studies including careful taking of history from family members. On the other hand, after electroshock treatment, the reverse is frequently true. Not only do many more patients thus treated return to the status of their former "own best self," but many patients report that they are even much better

than they had been for years prior to the overt onset of the depressive episode. Their spouses frequently say that the husband or wife respectively is not only better than he or she had been before the illness, but actually as well as he or she used to be many years ago. It is thus in retrospect that we can frequently establish the fact that such apparently acute depressive episodes actually were preceded by prodromals in the form of a gradual waning of the zest for life, frequently extending over periods as long as four to eighteen years. This type of restoration of the old zest for life in such cases certainly does not come about as frequently or characteristically in patients with depression who recover spontaneously or in response to purely psychologic treatment. This point, of course, cannot be ascertained from most statistics, which are based on rather gross observations, such as the ability of the patient to remain outside the hospital, but rather from a thorough acquaintance with the total personality of the patients. Fortunately, however, a number of authors make a careful distinction

Table 17. From Mayer-Gross^{110a}

	TOTAL 168 DEPRESSIONS % ECT	TOTAL 256 DEPRESSIONS % CONTROLS
CR.....	69.0	34.8
SR.....	7.7	33.2
Home or hospital invalid.....	13.0	18.0
Died.....	8.3	14.0
Untraced.....	1.8	—

between complete recovery, social recovery, and other significant improvement. By analyzing the figures of those authors separately, we find that among depressed patients of manic-depressive type, 51.3 per cent of the total of significantly improved patients treated with electroshock attained complete recovery (Table 14), compared with only 31.9 per cent of the nonshock-treated patients in the same category (Table 10). This finding is in remarkable agreement with a recent study by Mayer-Gross (not included in our figures, but given separately [Table 17] for purposes of comparison).

Mayer-Gross^{110a} also made a careful distinction between complete and social remission. While his combined complete and social recovery rate was 68 per cent in the group receiving only psychologic treatment—which is only slightly lower than 76.7 per cent for the patients treated with electroshock, the breakdown of these cases into those completely recovered and those only socially recovered showed that 69 per cent of the electroshock treated patients had attained complete recovery, and an additional 7.7 per cent social recovery. Among the controls treated with purely psychologic methods, only 34.8 per cent achieved full recovery, while an additional 33.2 per cent achieved a state of social recovery (Table 17).

Failures, Mental-Emotional Complications and Side Effects of Convulsive Electroshock Treatment. Nevertheless, the fact remains that even among depressions, statistically by far the most favorable illness responding to convulsive electroshock treatments, 16 per cent to 23 per cent failed to improve at all, and 31 per cent to 55.3 per cent failed to achieve full recovery. It behooves us to investigate what are the causes of these failures

and, by implication, what are the insufficiencies or disadvantages of convulsive electroshock treatment. The main causes for these failures are: First of all, convulsive electroshock does not relieve anxiety; anxiety, however, is not infrequently a complication of fundamentally depressive illnesses. Furthermore, anxiety not only remains unrelieved, but frequently becomes aggravated by convulsive electroshock treatment. Secondly, convulsive electroshock—usually temporarily, but sometimes over longer periods of time—reduces spontaneous activity and spontaneity in general. The latter occurs for the most part only in those cases in which there is also a build-up of anxiety. Thirdly, electroshock reduces awareness for memory contents. This again is frequently part of a state of general inhibition of thinking and acting which is also frequently associated with anxiety either primarily or secondarily. It is fortunate that recently an antidote for all three of these complications has become available, namely, nonconvulsive treatment, which will be discussed in the next section. At this point, we shall discuss the precise nature of these psychologic complications and resulting disturbances.

The only authors who give data concerning their statistical incidence are Deshaies and Pellier.⁵⁵ They report that troublesome memory disturbances occurred in 30 per cent of the patients under study, marked inhibition of spontaneity in 10 per cent. Disturbing build-up of anxiety ranging from an anxious neurasthenic syndrome (20 per cent) to states of more severe eruption of anxious agitation (3 per cent), and panicky excitement (4 per cent), complicated by marked mental confusion (5 per cent), occurred in a total of 32 per cent of cases. The total group with untoward mental complications, because of a slight overlap in the two main groups characterized by memory disturbance on the one hand, and overt anxiety on the other, comprises 40 per cent. This indeed correlates well with the number of cases that remained either unrelieved or incompletely relieved after convulsive electroshock treatment in our case material.

MEMORY DISTURBANCES. It is obvious that in memory disturbances following electroshock treatment we are dealing with two phenomena which, however, may frequently overlap or be superimposed upon each other. One of them is clearly nothing but the activation of the defensive operation known as isolation, sometimes with elements of denial. This applies to the selective forgetting of traumatic events. Holland⁸⁹ was the first to emphasize that the selectivity of this type of forgetting following electroshock has all the earmarks of an active repressive process on a psychologic plane, as opposed to a pathophysiologic condition. The other form of forgetting consists in a more generalized disturbance of awareness for memory contents which may be lacunar or spotty and may apply to both recent and old memory contents. More frequent, however, is the organoid pattern of forgetting of recent events, including retrograde and anterograde amnesia. Retrograde amnesia may sometimes go back as long as a year, depending upon the duration of the illness, thus undergoing a blending with the tendency to repress. Very frequently the retrograde amnesia coincides with the onset of the illness; in those cases it is obvious that the amnesic picture is a combination of a loss of awareness for memory contents with actual repressive operations. This is in contrast to the specific purely organoid form of retrograde amnesia, which goes back

only a few minutes prior to the shock treatment. The acquisition of new memory contents is sometimes similarly disturbed for a time after shock treatment in an anterograde manner. The most disturbing complaint is that of inability to remember familiar learned material such as cooking recipes in the case of a housewife, or mathematical or chemical formulae in the case of an engineer. This type of disturbance may last for a year or more after a series of electroshock treatments, although fortunately in most cases the duration is far less. This kind of memory defect is the most disturbing to the patient and is usually combined with a good deal of anxiety as well as disturbance of spontaneity. This type of memory disturbance differs from the ordinary organic memory disturbances occurring in cerebral disease or injury in that the patients are most painfully aware of their memory disturbance, while in characteristic "organic" memory disturbance this is usually not the case. This may, however, be due to the fact that judgment and other personality functions are not disturbed in our post-electroshock cases while they may be disturbed in patients suffering from organic memory defects due to cerebral disease or injury, although that is not always and necessarily the case.

There is no doubt that the original confusion and disturbed awareness for memory contents immediately after a shock treatment is "organic" of a sort, but the fact emerges with increasing clarity that this organicity does not imply an irreversible or even any other morphologic lesion, but rather suggests a physiologic inhibition of organic activity, possibly in the sense of Pavlov's inhibition, or along the lines of the Wedensky^{146, 147} effect. The main proof of organicity in any sense of the word is the fact that its incidence, intensity, and duration is correlated with the intensity of the electric currents used (Lemere¹⁰⁴). Pacella,¹²¹ however, emphasized that even this early memory disturbance is somewhat lacunar and spotty in type, thus differing from the more clearly retrograde and anterograde patterns observed after structural lesions. Diethelm⁵⁶ characterized the post-electroshock memory disturbance as a disturbance of correlation of isolated items in the integration of the whole picture (Gestalt). Masserman,¹⁰⁹ in animal experiments, proved it to consist in a disruption of complex patterns of both the neurotic and the normal behavior of the animals. Worchel and Narciso¹⁵² characterized the memory decrement following electroconvulsive shock as due to suppression of recently formed pathways; of particular interest is their finding that relearning was not affected. On the other hand, Gellhorn⁷⁷ proved that the problem is much more complex than that; he demonstrated that electroshock as well as insulin coma may bring about recovery of inhibited conditioned reactions. He advanced as a possible reason for this phenomenon that in electrically induced convulsions as well as in insulin coma the hypothalamic discharges to the cortex are increased, and thus may make subthreshold cortical processes supraliminal. By the same token, however, it is of course also possible that such discharges, if of sufficient strength, may inhibit cortical activity.

The fact that the memory disturbances following convulsive electroshock are not due to a structural type of lesion, but rather to a physiologic state of cortical inhibition, was proved most convincingly by the demonstration that these memory disturbances can be readily cleared up by nonconvulsive treatment which produces excitation of the cerebral cortex

in contrast to convulsive electroshock which appears to produce inhibition (Alexander²⁸). I have therefore come to regard the organoid memory disturbances which follow convulsive electroshock treatments as a decrease of awareness for memory contents rather than as true memory loss. The subsequent nonconvulsive stimulation that relieves this memory disturbance obviously acts as a stimulus that counteracts the inhibition of awareness produced by the convulsive electroshocks. A. C. Ivy⁹⁵ stated that, "learning and awareness are mutually dependent, because one can test for awareness only through learning or memory . . . Consciousness appears to be the result of adequate excitation of the cerebral cortex primarily, of such intensity as to attract one's attention." It appears that nonconvulsive electric stimulation produces such excitation of the cerebral cortex, in contrast to convulsive electroshock, which appears to produce inhibition. It thus appears that the decreased awareness for memory contents after convulsive electroshock is simply part of a larger syndrome of cortical inhibition manifested by decrease of spontaneous activity, by decrease in awareness for memory contents, as well as by increase in anxiety. Anxiety has been found to occur as a reaction to impaired activity of the cortex, for instance, in certain forms of intoxication such as tetraethyl lead poisoning.^{83, 98, 27} There is no evidence at all suggesting that the memory disturbance following electroshock is due to a structural lesion in the ordinary sense. Such a lesion certainly could not be as readily reversible by nonconvulsive treatment as this memory disturbance has proved itself to be.

Those observers who assumed the cause of the memory disturbance to be the result of an organic structural damage have never succeeded in proving their point. For example, Brody⁴² considered prolonged memory defect following convulsive electroshock therapy as implying permanent or semi-permanent damage to the brain in the sense of a structural lesion. Careful study of Brody's cases, however, shows that they do not prove his point. First of all, 4 of the 5 cases in which Brody reported prolonged memory defect following electroshock treatment, which he considers due to organic impairment, are cases complicated by anxiety. It is of particular interest to find that the characteristics of these memory defects are by no means those which are found in organic memory defect. The disturbance mainly affected long-known familiar material. The inability to recall names of persons and places and habits of work including routine items is more characteristic of *feelings of estrangement than of memory loss*. Also the marked subjective awareness of this loss and the anxious elaboration and strain with which the patients reacted to it are likewise more characteristic of anxious preoccupation than of organic memory defect. Confirmatory of this impression is the fact that only one of Brody's 5 patients is a manic-depressive, and even she has obvious reactions of apprehension to the treatment and therefore very likely is a patient in whom anxious "neurotic" reactivity played a significant part. The fact that she became markedly apprehensive after a few electroshock treatments—that she thought the treatments were making her worse—certainly suggests that this patient was suffering from epinephrine-precipitable anxiety of the neurotic type rather than from pure depression. It thus becomes obvious that what the author described as memory defect is more likely inhibition of awareness associated with general *anxious inhibition* brought about by the anxiety-

increasing and cerebation-inhibiting effects of convulsive electroshock therapy.

Bodamer³⁹ likewise considered the memory disturbance following electroshock treatment as "organic," although the 4 cases he reported rather indicate the contrary. In 2 of his cases, the retrograde amnesia neatly coincided with the onset of the psychosis; in the other 2 with the stressful event which precipitated the psychosis (in one case the funeral of a close friend, in the other case the expulsion from her home).

CHANGES IN CEREBRAL ELECTROACTIVITY. Electroencephalographic studies likewise failed to produce evidence of irreversible damage to brain tissue, the findings being consistent with temporary inhibition of normal cortical activity, and the appearance of abnormal rhythms, particularly slow high voltage activity, similar to those produced by other cortex-disorganizing agents, such as anoxia, barbiturate narcosis, ether anesthesia, hypoglycemia, or shock from exposure to cold.

The disturbance of cortical electroactivity after convulsive electroshock treatment is always temporary. It lasts a far shorter time than the memory disturbance, even in patients who have been treated with as many as four major seizures daily for seven consecutive days (Callaway and Boucher⁴³). It consists essentially in hypersynchrony manifested by slow wave activity. This slow activity increases strikingly following termination of treatment but soon decreases and finally disappears in from one to three weeks after treatment (Bagchi et al.,³¹ Schwab¹³⁸). Five months after termination of treatment no residual slow activity persists.⁴³ Proctor and Goodwin¹²⁴ showed that patients achieving social remission showed less slow wave activity than the patients still requiring continued hospital care. This difference was particularly marked in those cases who were treated by unidirectional current, while in the group receiving treatment by alternating current this difference was obscured by the more marked and general slow wave activity produced by the alternating current. The authors were not able to correlate the degree of slow activity with memory disturbance; they emphasized that the memory disturbance following electroshock is a more complex phenomenon than one testable by the usual simple memory tests.

PSYCHOSOMATIC INHIBITION. Inhibition of neurophysiologic activity can also be discerned in the neuro-endocrine field. There is usually some degree of amenorrhea in women following electric shock treatment, its duration roughly correlated with the number of electroshocks administered (Liepelt¹⁰⁶). In men there is temporary impotency or decrease of potency which may last for two to ten weeks following a series of electroshock treatments. It is not sufficiently accounted for by depression of endocrine functions per se, especially those of the adrenal cortex or the gonads (Michael¹¹²), but rather appears to be a manifestation of a temporary inhibited state of sexual appetite, paralleling the general inhibition of spontaneity which frequently outlasts a series of convulsive electroshock treatments for about that time. Such patients may complain that although their depression is completely relieved, they still do not experience their usual old zest. Some may complain that their head feels empty or heavy; others complain that they feel as if they were not really quite alive. These subjective feelings and the associated loss of spontaneity gradually fade into the more obvious expressions of anxiety, especially the states of

anxious preoccupation which Deshaies⁵⁵ classified as "neurasthenic complications" of electroshock treatment.

EXACERBATION OF ANXIETY. Build-up of anxiety is indeed the most important psychophysiologic complication of convulsive electroshock treatment. It occurs in spite of successful relief of depression and of whatever other regressive psychotic manifestations the patient had been suffering from originally. In these cases anxiety frequently increases in a reciprocal manner as depression lessens. In most cases this does not take place until after about the third or fourth treatment but sometimes this phenomenon may appear after the first treatment. This build-up of anxiety in its mildest form may consist merely in anxious aversion to treatment. Such patients may say, "I know these treatments have helped me and I know they have never hurt me, Doctor, but I have become mortally afraid of them." In conjunction with the fear of treatments other fears and anxious thoughts likewise may come to the surface, such as fear of going insane, fear of screaming and making a spectacle of oneself, fear of "losing control," with the implication of hurting or killing other people. In some patients this anxious build-up may assume the form of a purely neurasthenic syndrome as described by Deshaies.⁵⁵ In 2 of my patients this was associated with tachycardia, orthostatic hypotension, and exercise intolerance, characteristic of the syndrome of neurocirculatory asthenia. Neither of these two patients had ever suffered from similar complaints before treatment. Other patients may develop more serious states of anxious agitation with constant fears of impending disaster frequently keeping them wakeful at night or waking them out of a sound sleep.

Flescher⁶⁸ emphasized that anxious resentment and associated aggressive reactions in these patients interfere with and destroy the otherwise beneficial effects of convulsive electroshock treatment. Chodoff, Legault, and Freeman⁴⁶ pointed out that for this reason electroshock is relatively ineffective in anxiety states and in patients with obsessive-compulsive symptomatology and that it may be responsible for the poor results of electroshock treatment in most schizophrenic psychoses. Rondepierre¹³² pointed out that the increase of anxiety may aggravate the disease in some schizophrenics. When build-up of anxiety becomes combined with organoid postshock confusion, states of wild panicky agitation with maniacal deliriod excitement and impulsive, uncontrollable aggressive and destructive outbursts may ensue, which if untreated may last for days and weeks. Although this picture had been observed sporadically before, it was first carefully described by Frosch, Impastato and their coworkers,⁷⁰ who pointed out that these reactions were more severe than the usual organoid confusion, anxious subjective reactions to memory difficulties and confusion being more pronounced. A common picture in these confused psychotic reactions was that the patients were disturbed, agitated, restless, assaultive, destructive, sometimes suspicious and paranoid, only occasionally stuporous, the picture profoundly differing from the clinical picture of the psychotic episode prior to electroshock treatment which was a purely depressive picture in the majority of the cases. Each of the patients had received more than ten electroshock treatments and they had been treated daily, which raised the point that this may have been excessive. However, I have seen such syndromes arise also in patients who had been treated entirely with standard doses of electroshock treatment. In the cases of my observation,

however, there had been admixture of anxiety or mild paranoid trends prior to electroshock treatments.

Prognostic Criteria for Good Therapeutic Response to Convulsive Electroshock Treatment. The problem of avoidance of mental complications of the various types outlined above brings up the important problem of prognostic criteria, especially in nondepressive groups in which recovery rates are lower and more variable. Such prognostic criteria have been evolving slowly. Gold and Chiarello⁷⁹ believe that depressive features, suicidal intent, muteness, and confusion are more likely to be benefited by electroshock than are illnesses characterized by loss of interest, negativism, and grandiose delusions. (By implication, it is obvious that these two groups are characterized by breakdown of defenses in the group responding favorably to electroshock treatment, and by florid activity of defenses in the group not responding to electroshock treatment.)

Clark, Kiefer, and Gerson⁴⁷ believe that the reaction to sodium amytal is able to presage good response to electroshock, especially when temporary improvement in behavior, speech, mood, and insight are considered. This correlation is not only of practical, but also of theoretical interest in view of the basic physiologic event which these two procedures have in common: namely, release or stimulation of the brain stem respectively. Sodium amytal narcosis brings about temporary improvement by cortical sedation and release of the brain stem; the physiologic component most essential for the more profound and lasting antidepressant effect of convulsive electroshock is its tonic phase, brought about by excitation of the brain stem, while the clonic (cortical) phase is not essential (see Chapter IX).

Scherer¹³⁶ believes that good scores with categorical thinking tests, rate of manipulation and memory efficiency tests presage a favorable outcome. This psychologic finding also has its physiologic correlation: Funkenstein and Meadow (ref. 39, p. 31) have demonstrated a positive correlation between disturbance in categorical thinking and the presence of anxiety maintained by norepinephrine. Such anxiety is notoriously refractory to treatment, especially to treatment by electroshock.

THE PROGNOSTIC VALUE OF THE PSYCHOSOMATIC RESPONSE PATTERN (FUNKENSTEIN TEST). The most important practical means, however, of predicting response to electroshock is the Funkenstein test,⁷¹⁻⁷⁵ which as described above consists of two phases. The first phase is the injection of 0.025 mg. of epinephrine intravenously. If such an injection evokes anxiety, troublesome anxiety is bound to erupt during electroshock treatment. This is due to the fact that the most important physiologic effect of electroshock treatment is a lowering of basic sympathetic tension associated with enhanced responsiveness to adrenergic stimuli. If adrenergic stimulation had evoked anxiety before electroshock treatment, this reaction to epinephrine will become enhanced after electroshock, resulting in an increase in epinephrine-precipitable anxiety. In such cases, electroshock is contraindicated unless other measures are taken to prevent build-up of epinephrine-precipitable anxiety, among which nonconvulsive treatment and insulin are the most helpful.

The second phase of the test is the intramuscular injection of 10 mg. of mechoyl. If this injection leads to a pronounced and prolonged fall in blood pressure, irrespective of whether this fall is associated with the

precipitation or the relief of symptoms, the illness is one that will react favorably to electroshock treatment. On the other hand, if mecholyl fails to lower blood pressure, presumably elevated by enhanced sympathetic tension, electroshock treatment will not bring about significant or lasting relief. This is due to the fact that elevated blood pressure irreducible by mecholyl is maintained not by epinephrine, which can be neutralized by mecholyl, but by norepinephrine, which cannot be neutralized by mecholyl. Enhanced sympathetic tension mediated by norepinephrine is not amenable to modification by electroshock treatment. It is, however, amenable to insulin and sometimes, in the rare menopausal depressions, to gonadotropic substances, such as progynon B. On the psychosomatic level, the reaction to norepinephrine is similar to the psychosomatic reaction to anger, the reaction to epinephrine similar to the reaction to fear (A. Ax³⁰).

Electroshock treatment, viewed neurophysiologically, appears to be a strong cholinergic stimulus which succeeds in lowering sympathetic tension, provided the sympathetic tension was transmitted by epinephrine. After successful treatment the resulting lowered sympathetic tension is associated with a lessened reactivity to parasympathetic stimuli: the blood pressure then will drop only slightly below the base line after an injection of mecholyl and will return to the base line within 8 to 10 minutes. The response to sympathetic stimuli, however, will become greater. In patients free from epinephrine-precipitable anxiety, the enhanced responsiveness to epinephrine after treatment will have no untoward subjective or objective effects in the vast majority of cases. Cases in which anxiety was precipitated by epinephrine for the first time after treatment are rare. I have observed only one such case. If, however, anxiety was already precipitable by epinephrine before treatment, this anxiety is bound to become enhanced after treatment. Such build-up of anxiety will invariably disturb the progress of treatment, particularly in out-patient cases but also in hospitalized patients. It frequently becomes so alarming that electroshock treatment must be cut short. Furthermore, even if reasonably good results in regard to relief from depression or other psychotic symptoms are obtained in spite of the build-up of anxiety, the persistence of epinephrine-precipitable anxiety after treatment will remain a stress factor leading sooner or later to full relapse, the patient subjectively and clinically being finally worse than before treatment. I should like to give two characteristic examples:

Figure 50A shows the results of the pretreatment Funkenstein test of a patient falling into the group responding well to electroshock therapy. There was prolonged lowering of blood pressure on injection of mecholyl and a normal reaction to the injection of epinephrine without precipitation of anxiety (Fig. 50A). After a series of electroshock treatments and clinical recovery, the basic sympathetic tension had become relaxed as indicated by a lowering of basal blood pressure (Fig. 50B, showing the results of the post-treatment Funkenstein test of the same patient). The reaction to mecholyl had become normal in that the blood pressure fell and returned to its preinjection level within 10 minutes. The reaction to epinephrine had become enhanced, but no anxiety was precipitated by it (Fig. 50 B). This patient achieved a sustained good therapeutic result from electroshock, as do 90.9 per cent of all patients showing this type of pretreatment autonomic response pattern (Funkenstein et al.⁷⁵).

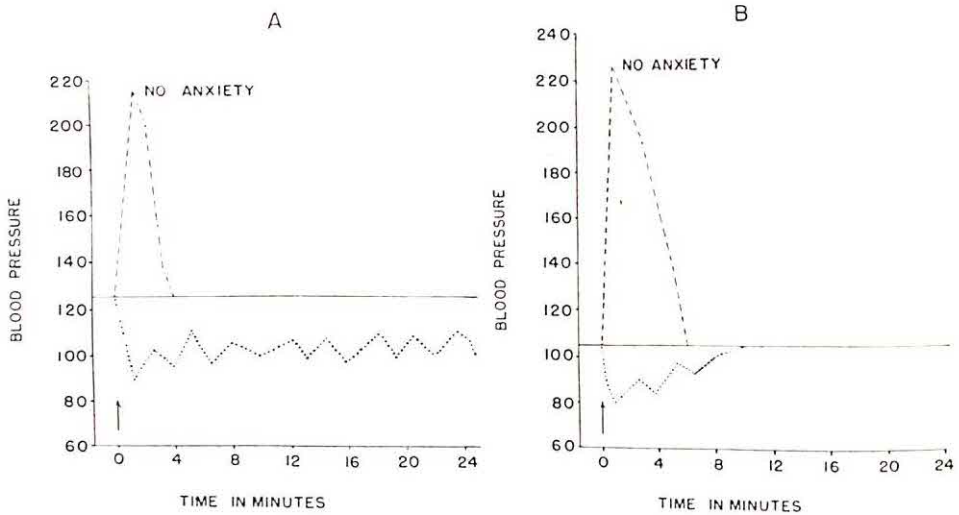


Fig. 50. Male, aged 34 years, suffering from schizophrenic psychosis, who recovered after convulsive electroshock therapy.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before treatment. Note marked and prolonged fall of blood pressure after injection of mecholyl, and absence of epinephrine-precipitable anxiety.

B. Funkenstein test after electroshock therapy and clinical recovery. Note normal slight transitory fall of blood pressure after injection of mecholyl; enhanced response to epinephrine, but without precipitation of anxiety. (From Funkenstein, Greenblatt, and Solomon: *Am. J. Psychiat.*, vol. 106, 1950.)

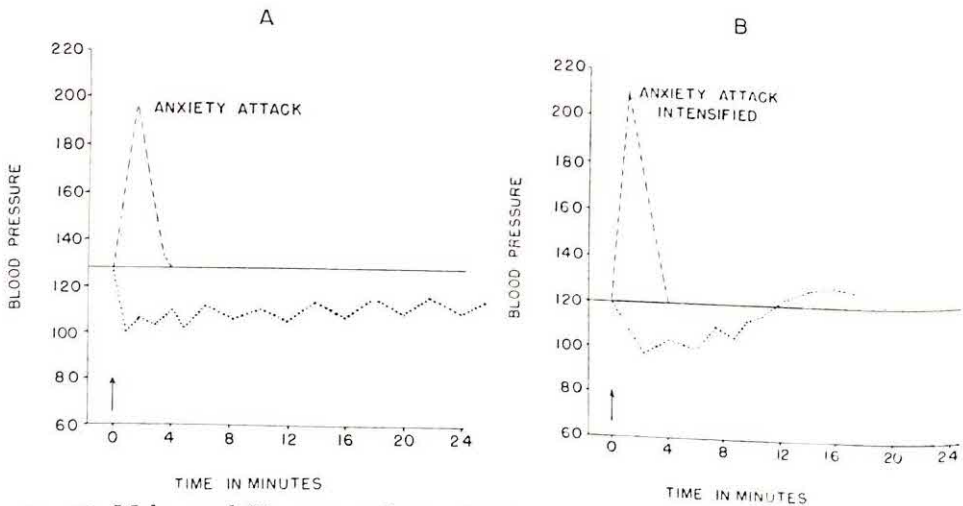


Fig. 51. Male, aged 28 years, suffering from schizophrenic psychosis; exacerbation of anxiety after 6 convulsive electroshock treatments.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before treatment. Note marked and prolonged fall of blood pressure after injection of mecholyl similar to that shown in Fig. 50 A, but anxiety attack precipitated by injection of epinephrine.

B. Funkenstein test after 6 electroshock treatments. Note increase in epinephrine-precipitable anxiety. (From Funkenstein, Greenblatt, and Solomon: *Am. J. Psychiat.*, vol. 106, 1950.)

Figure 51 A shows the results of the pretreatment Funkenstein test of a patient whose reaction to mecholyl prior to electroshock treatment was similar to that in Figure 50 A in that there also was a pronounced and prolonged fall of blood pressure. Thus, as far as the reaction to parasympathetic stimulation was concerned, a good therapeutic response to electroshock treatment could be anticipated. Epinephrine injection, however, produced an anxiety attack, predicting an unfavorable reaction to electroshock in terms of build-up of postshock anxiety. After electroshock treatment (Fig. 51 B), the response to mecholyl improved as predicted in that the blood pressure fell and returned to the preinjection level within eleven minutes, followed by slight overshooting above the preinjection level. The basic sympathetic tension had likewise responded in that the resting blood pressure was now lower than before treatment. However, the anxiety attack precipitable by injection of epinephrine had become intensified and the patient, although relieved from his major psychotic symptoms, had developed severe postelectroshock anxiety which continued for more than a year after treatment.⁷³ Many such patients relapse or become worse because the added or enhanced overt anxiety not only may bring about a relapse of the original condition but may actually make it worse, because of the added or enhanced element of anxiety. For this reason, only 36.4 per cent of all patients showing epinephrine-precipitable anxiety with the Funkenstein test achieve social recovery, which is less than the remission rate expected without physical treatment. Ninety per cent of these patients, however, do well with insulin treatment (Funkenstein et al.⁷⁵) or with combined convulsive-nonconvulsive electric treatment, which will be discussed further below. It is obvious that patients with epinephrine-precipitable anxiety should not be treated by convulsive electroshock unless other physical measures, in addition to psychotherapeutic management of the patient, are taken to control the eruption of anxiety. Among these measures nonconvulsive electric treatment, combined convulsive-nonconvulsive treatment, and insulin treatment (subcoma or coma) are the most effective.

Nonconvulsive Electric Treatment. Nonconvulsive electric treatment relieves anxiety, increases spontaneous activity, and restores awareness for memory contents, irrespective of the origin of disturbances in these areas; that is, whether or not they occurred spontaneously—singly or together—in states of perplexed agitation or were precipitated by convulsive electroshock treatment.

The Psychophysiologic Effect of Nonconvulsive Electric Treatment on Abnormal Mental States. Associated with the relief of anxiety brought about by nonconvulsive electric treatment is a marked softening and lowering of previously rigid and frantic defenses. Obsessions and compulsions, in particular, at first lose their urgent quality, and as treatments continue, may vanish altogether. Rapport becomes enhanced, verbalizations and insight are facilitated, and the patient becomes remarkably accessible to interpretations. It is therefore important that this treatment always be given in association with ventilative and interpretive psychotherapy. But as anxiety is relieved and defenses are abandoned or give way following nonconvulsive treatment, depression, if coexistent, may become enhanced or if previously latent may become overt, in the same reciprocal manner in which anxiety may erupt after relief of depression following convulsive

treatment. In these cases the physician must skillfully steer the patients through the Scylla of anxiety and the Charybdis of depression by giving nonconvulsive treatment whenever anxiety is predominant, or convulsive treatment whenever depression comes to the fore.

The Clinical Effects of Nonconvulsive Treatment in Mental Diseases. The chief groups of conditions in which nonconvulsive treatment alone is helpful without recourse to other forms of physical treatment are certain acute schizo-affective panics and certain so-called reactive depressions in which the symptomatology is chiefly one of anxious agitation. In the latter group frequently one or two treatments, at most seven, may restore the patient to his former equanimity without rebounding or build-up of the slight element of actual depression that may have been associated with the illness. This has its counterpart in the nuclear group of classical endogenous depressions in which anxiety likewise does not rebound after convulsive treatment.

Certain schizoaffective panics are equally promptly responsive to nonconvulsive treatment. A characteristic example is the case of a 26 year old recently married veteran who on a short journey away from his home was suddenly overwhelmed by the conviction that all women had become repulsive to him, which aroused his fear that he suddenly might have become homosexual. He was entirely unresponsive to psychotherapy, and his condition was rapidly deepening into an acute catatonic state. After two nonconvulsive treatments he was completely relieved of his fear and responded superbly to insight therapy. The minor marital psychosexual maladjustment in which his young wife's attitude figured prominently was subsequently adjusted in joint therapy with patient and wife. The total therapy comprised two nonconvulsive electric treatments, seven psychotherapy sessions with the husband and three psychotherapy sessions with the wife, extending over a total of seventeen days. I am certain that the patient would have been slated for a much longer siege of illness if physical treatment had not been used, and he would have responded unfavorably in terms of build-up of anxiety if he had been subjected to convulsive electroshock treatment.

Another group of conditions in which nonconvulsive electric treatment can be of decisive therapeutic benefit are the obsessive-compulsive states, not only the acute states which are akin to schizoaffective panics but also compulsive obsessive states of remarkable chronicity, such as hand washing compulsions, obsessive cancerphobias, and obsessive fears of committing crimes, that have been of remarkably long duration, up to eight years in my clinical experience. As the obsessions and compulsions become relieved or vanish altogether in the course of nonconvulsive treatment, depression usually comes to the fore, associated with a corresponding change in the autonomic response pattern. These residual depressive states then respond to convulsive or combined convulsive-nonconvulsive electric treatment. This supplemental interaction of the two forms of treatment leads us to consider the methods of integration of nonconvulsive treatment with convulsive treatment.

Integration of Nonconvulsive with Convulsive Treatment—Nonconvulsive Treatment as the Opening Phase of an Electric Treatment Series. In cases in which anxiety as well as depression, latent or overt, is present, nonconvulsive treatment is useful in toning down anxiety at the beginning

of the electric treatment series, provided the physician is ready to change to convulsive electric treatment or combined convulsive-nonconvulsive treatment as soon as the anxiety is relieved and depression comes to the fore.

It is important not to wait too long for such a revision of the course, especially at the beginning of the treatment series when the rapport between physician, patient, and family is still in the developmental phase. The importance of this flexibility was vividly brought home to me by the case of a 32 year old woman who consulted me for an anxiety tension state with schizoid-obsessive features of nine years' duration, severe for three years, which proved unamenable to psychotherapy and therefore presented indication for physical treatment. One nonconvulsive treatment elicited a profuse release of desperate crying, abject despondency, and self-accusations. The patient and her mother were so appalled by this result that they refused all further physical treatment—a decision which neither I nor the referring physician was able to change. The patient remained in a severe state of depression for three months and returned to her former anxiety tension state only very slowly after intensive psychotherapy in other hands. I have since made it a rule whenever lachrymose depressive outbursts continue for more than an hour after nonconvulsive treatment to administer convulsive or combined treatment forthwith.

It is well, however, in such cases in which anxiety as well as depression plays a role to begin always with nonconvulsive treatment, since it is better for the rapport with the patient to begin with the relief of anxiety and with a form of treatment which does not cut the thread of awareness, since many patients, especially those with obsessive personality trends, regard this as the most threatening and unpleasant aspect of convulsive electroshock treatment. The reason why nonconvulsive treatment does not cut the thread of awareness is difficult to explain, especially since an anesthetic is usually used with nonconvulsive treatment. However, patients wake up and are able to continue a conversation right where they had left it off before the treatment, and they wake up fully oriented, which is never the case with convulsive treatment. More about this will be said in discussing the dynamics of the immediate post-treatment phase (Chapter XV, p. 399ff.). Be this as it may, anxious patients usually wake up from nonconvulsive treatment relaxed and happily relieved (with the exception of occasional cases in which it may precipitate depressive outbursts), and they regard the treatment as pleasant, while convulsive treatment is usually regarded as unpleasant by the patient even if it has helped him.

By beginning with nonconvulsive treatment, it is thus possible to condition patients gently to shock treatment who, if shocked without such preparation, might have responded with aversion and build-up of anxiety. For this reason this method also allows greater use of psychotherapy in the course of the treatment than is possible—especially at the outset—if only convulsive electroshocks are given.

The Relief of Exacerbations of Anxiety after Convulsive Electroshock. When build-up of anxiety develops during or at the end of a convulsive electric treatment series, a few nonconvulsive treatments relieve the build-up of anxiety promptly. If this can be accomplished by a few treatments, usually no re-exacerbation of the depression will ensue, provided the depression had been treated by sufficient amount of preceding convulsive

treatment. A woman aged 35 years developed a severe state of anxious agitation after five combined convulsive-nonconvulsive treatments which had been administered to treat her severe depression complicated by epinephrine-precipitable anxiety. Following the relief of her depression two nonconvulsive treatments relieved the eruption of anxiety and made it possible for her to gain further improvement by working out her underlying personality problem in psychotherapy, and to achieve first a workable social recovery, finally complete clinical recovery. Nonconvulsive treatment is also remarkably effective in relieving the severe states of anxious agitation combined with maniacal deliriod confusion with impulsive aggressivity of the type described by Frosch and Impastato.⁷⁰

A 45 year old man received nineteen electroshock treatments between December 28, 1949, and February 3, 1950, for the relief of a depression with mild paranoid trends. Seven days later, on February 10, 1950, he developed a severe deliriod maniacal state of excitement. Loudspeakers droned obscene epithets at him from all sides as well as through the radiators of the heating system emanating from people massing at the hospital door and intent on "getting" him. He shook in fear and anger; he would tremblingly hurl himself against anybody who entered his room; he ripped off his clothes because they had been polluted by other people's sperm that had been smeared all over them. His state of excitement which was so unmanageable that a staff member wondered whether a lobotomy might not become advisable, continued unabated for 15 days. One nonconvulsive treatment had a remarkably calming effect and after a total of five nonconvulsive treatments given between February 25 and March 3, 1950, the excitement subsided. Improvement of the original psychosis progressed gradually, and six weeks later the patient was discharged recovered not only from the acute deliriod post-electroshock excitement but also from his depression and underlying paranoid trends, and returned to his work.

A similar case is that of a depression with marked retardation in a young girl aged 19, an observation of Dr. Charles Saltzman at Bournewood Hospital, who developed a manic state after four convulsive electric treatments, then after eight more convulsive electroshock treatments were given, a delirious panicky state of terror resulted which intensified further when four more convulsive treatments were given. For ten days she shrieked incessantly at the top of her voice and had to be restrained because she would hurl herself against the windows in blind terror whenever released. After two nonconvulsive treatments this girl achieved a complete state of recovery.

Nonconvulsive treatment is a therapeutic measure of decisive clinical importance in the control of these deliriod reactions, which are probably the result of combination of the original anxious, paranoid, depressive or manic psychosis with post-electroshock anxiety and organoid post-electroshock confusion. Nonconvulsive treatment in these cases obviously relieves both the anxiety as well as the confusional state. Both of these states may be related to inhibition of cortical activity left in the wake of convulsive electroshock.

The Relief of Memory Disturbance after Convulsive Electroshock. Stimulation of inhibited cortical activity, a quite probable effect of nonconvulsive treatment, may also be the mechanism of one of the most remarkable effects of nonconvulsive treatments, namely that of restoration of dis-

turbed awareness for memory contents. I made this observation early in my work with nonconvulsive treatment²⁸ and have since found it confirmed in a great number of cases. A particularly spectacular case was that of a 44 year old electronics engineer whose postshock memory disturbance, which developed after eleven electroshock treatments administered for depression, was particularly annoying to him because it involved even simple data and formulas with which he had been familiar for years and which he constantly needed in his work. It continued unabated for four months and was promptly and dramatically relieved after the first of two nonconvulsive treatments. The patient later stated that immediately upon awakening from the first nonconvulsive treatment: "I became aware that my head was clearer than it had been for many months—the sense of being overtired and of the 'veil effect over my memory' were entirely gone. My eyes were clear. A sense of well being that I had not known for years was present. Upon leaving the hospital I walked two or three miles. My eyes were very good—no discomfort. That very afternoon when I went back to the lab, I found that my head was clear and that everything I knew came to my mind easily and readily." Another treatment was given two days later although the disturbance had not recurred. His recovery has remained sustained for the past three years.

A recent similar case was that of a 32 year old woman, whose post-electroshock memory disturbance outlasted a series of twenty-one convulsive treatments by five months. The first nonconvulsive treatment administered then gave her considerable relief. She said a week later: "I feel good—I seem to do things faster, and when I wake up in the morning I feel wide awake. That morning after you gave me that treatment I woke up feeling wide awake for the first time in months . . . It is hard to explain, but I feel a lot different. I remember things now that I did not remember before, and a lot of things that I had forgotten flashed back into my mind." Two more nonconvulsive treatments given at weekly intervals further increased her feeling of clarity of mind.

In cases of less severe memory disturbance the relief of the postshock memory disturbance by nonconvulsive treatment is less spectacular but equally definite. Relief of memory disturbance is noticeable also when nonconvulsive treatment is not given as a separate treatment but in the form of nonconvulsive countershock immediately after the convulsive treatment. Whenever the nonconvulsive countershock was given, I found not only that the memory disturbance following electroshock was less pronounced than with convulsive electroshock alone, but that, in a number of cases receiving convulsive electroshock only, memory frequently was dramatically restored as soon as a further convulsive treatment followed by a nonconvulsive countershock or a nonconvulsive treatment alone was given. Such treatments were particularly helpful at the end of the series.

The Relief of Inhibition of Spontaneity after Convulsive Electroshock. Equally dramatic is the relief from reduction of spontaneous activity which frequently outlasts the affective manifestations of depression following convulsive treatment. As soon as nonconvulsive countershock was added to the convulsive treatment, patients who had been content to sit inertly for days following convulsive treatment alone became animated, alert, eager to go for walks outdoors, and to take part in other occupational therapy activities. I therefore found nonconvulsive countershock highly desir-

able in depressed patients in whom inhibition and inertia had remained a problem after they appeared to have recovered from their depression.

The Reciprocal Nature of the Effects of Nonconvulsive and Convulsive Electric Treatments. It thus appears that nonconvulsive treatment relieves the most important psychopathologic symptom that is not only not accessible to but actually aggravated by convulsive electroshock therapy, namely anxiety, as well as all those additional conditions which appear as mental complications caused by convulsive electroshock, namely confusion and disturbance of awareness for memory contents including their combinations with anxiety culminating in the Frosch-Impastato syndrome.

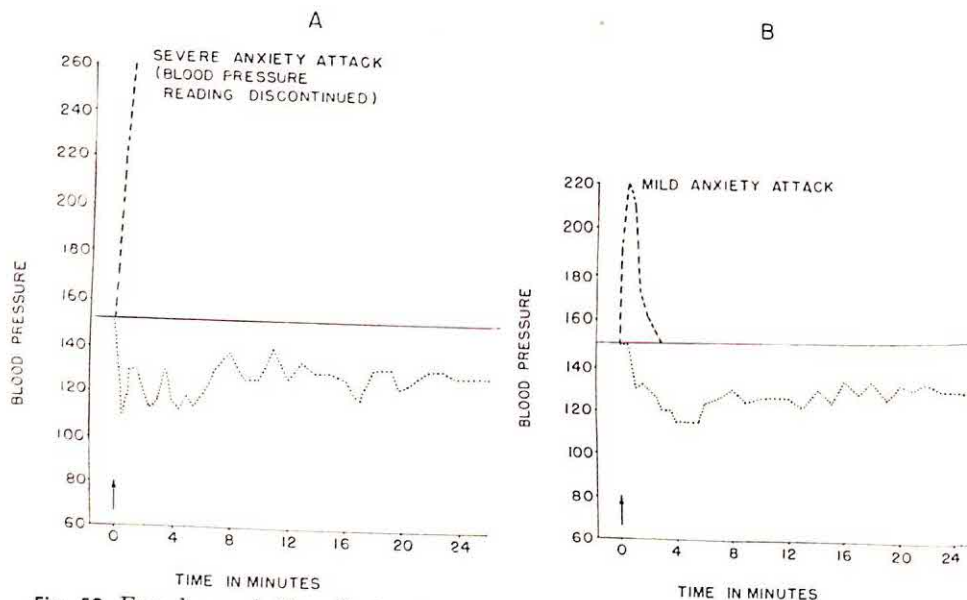


Fig. 52. Female, aged 42, suffering from manic psychosis with marked irritability. The broken line represents the blood pressure response to epinephrine (0.025 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).
A. Funkenstein test before treatment. Note greatly enhanced response to epinephrine with precipitation of severe anxiety attack as well as marked and prolonged fall of blood pressure following injection of mecholyl.
B. Funkenstein test after 9 nonconvulsive electric treatments administered between July 1 and 19, 1951. Note lessening of epinephrine-precipitable anxiety.

Changes in Psychosomatic Response Pattern Brought about by Nonconvulsive Electric Treatment. The response pattern of the autonomic nervous system brought about by nonconvulsive treatment as compared to convulsive shock treatment conforms to the reciprocal or antipodic position of the therapeutic action of nonconvulsive treatment and convulsive shock respectively.

Figure 52 A shows the results of the pretreatment Funkenstein test in the case of a 42 year old woman suffering from severe chronic manic-depressive psychosis of ten years' duration, who had been presenting the psychopathologic picture of a chronic irritable mania for the past five years. The Funkenstein test showed enhanced sympathetic tension manifested by elevation of basal blood pressure. There was marked and prolonged lowering of blood pressure on injection of mecholyl, as well as a greatly increased reaction to epinephrine

complicated by a severe anxiety attack. Nine nonconvulsive treatments relieved the severe anxiety and overreaction to epinephrine (Fig. 52 B). The basal

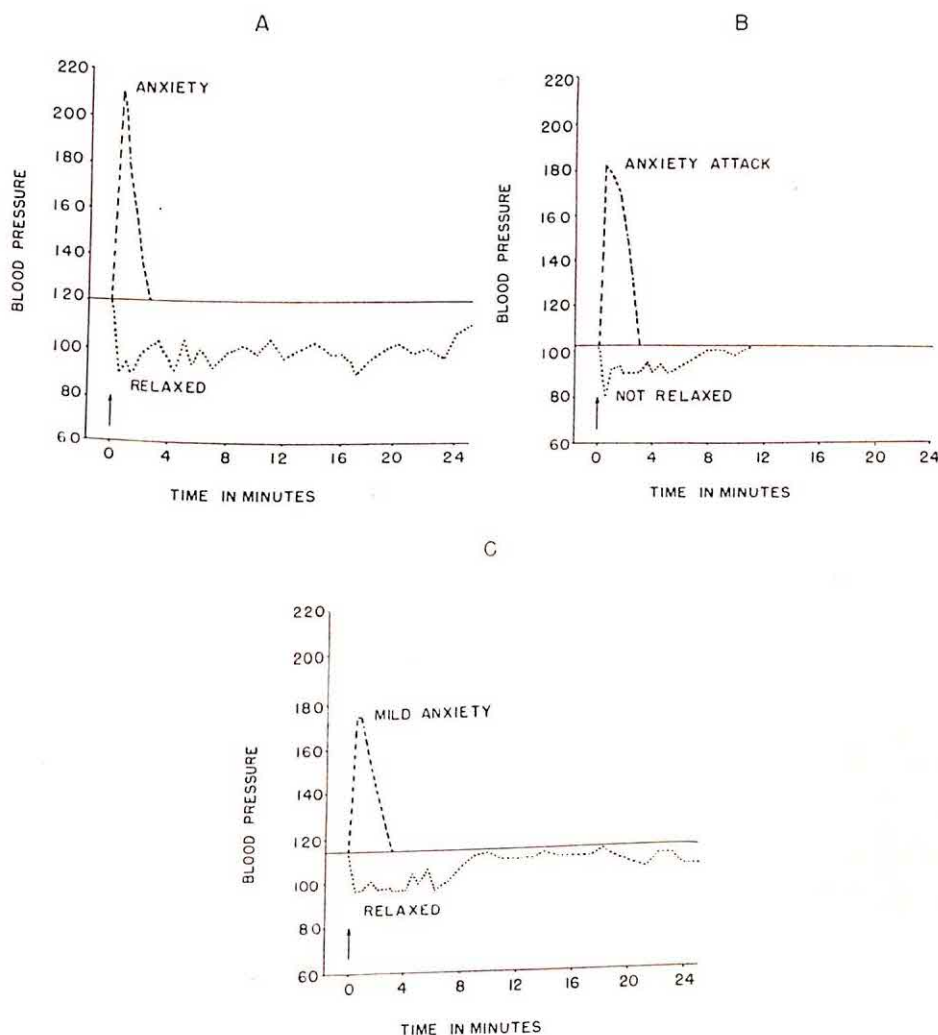


Fig. 53. Female, aged 35.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test prior to treatment for paranoid schizophrenic state with depressive features. Note marked and prolonged fall of blood pressure after injection of mecholyl, as well as epinephrine-precipitable anxiety. Patient recovered from this psychosis after 60 deep insulin comas.

B. Funkenstein test 1½ years later when anxiety reerupted without other psychotic manifestations. Note epinephrine-precipitable anxiety, but normal slight transitory fall of blood pressure following injection of mecholyl.

C. Funkenstein test after 17 nonconvulsive electric treatments which relieved anxiety but allowed depression to come to the fore. Note lessening of epinephrine-precipitable anxiety, but prolonged fall of blood pressure after injection of mecholyl.

blood pressure remained unaffected, as well as the marked and prolonged overreaction to mecholyl. Psychologically the irritable, hostile, compulsive aspects of her manic drive were relieved at this point, but the essential thymergasic

disturbance remained unabated. This patient finally achieved a complete recovery after subsequent convulsive electroshock treatments and insulin subcoma treatments followed by insulin coma treatments (see p. 241). The probable reason why in this case the basal blood pressure was not further elevated and the overreaction to mecholyl not further enhanced after nonconvulsive treatment was that these two conditions had already been maximal.

Another characteristic case in point is that of a 35 year old woman presenting the clinical picture of an anxiety neurosis with schizo-affective features, who one and one-half years before had recovered from a paranoid schizophrenic

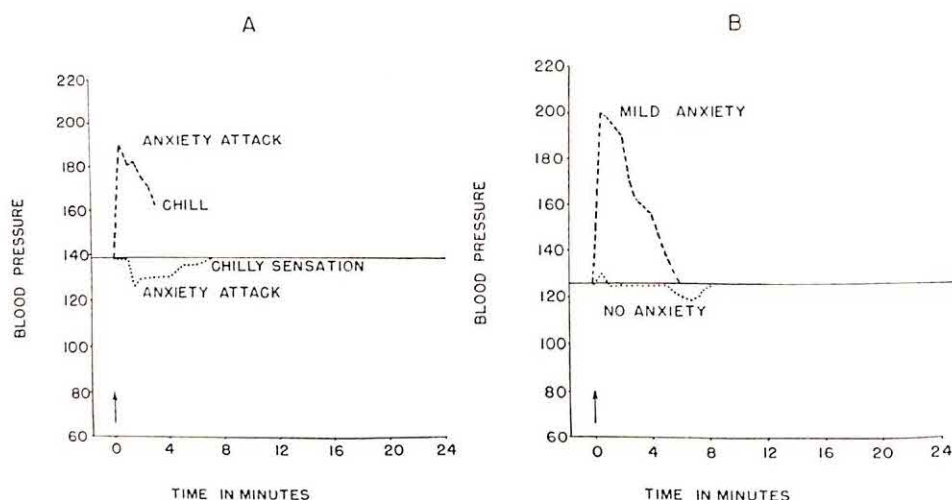


Fig. 54. Female, aged 45, suffering from agitated depression.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before treatment. Note anxiety attack and chill after injection of epinephrine as well as after injection of mecholyl.

B. Funkenstein test after series of electric treatments resulting in clinical recovery. The treatments consisted of a series of 4 nonconvulsive treatments followed by 4 combined convulsive-nonconvulsive treatments. Note normal response to mecholyl and marked lessening of epinephrine-precipitable anxiety.

state with depressive features with the aid of sixty deep insulin coma treatments after having remained unresponsive to two years of analytic psychotherapy. Before I had started her on insulin there was both prolonged overreaction to mecholyl as well as epinephrine-precipitable anxiety (Fig. 53 A). She achieved a full clinical recovery after sixty insulin comas. One and one-half years later, in her mild relapse, the reaction to mecholyl was within normal limits, but there was an anxiety attack on injection of epinephrine (Fig. 53 B). Seventeen nonconvulsive electric treatments greatly reduced the epinephrine precipitable anxiety (Fig. 53 C), but slight depression came to the fore associated with increase in basal blood pressure and prolonged over-reaction to injection of mecholyl in terms of moderate but prolonged drop of blood pressure below the preinjection level (Fig. 53 C). Subsequent convulsive treatment greatly enhanced the anxiety in spite of the fact that it was given combined with further nonconvulsive treatment. The patient, however, achieved an eventual state of significant improvement bordering on recovery, after insulin subcoma treatment combined with a few short and light insulin comas. Subsequent psychotherapy brought about complete clinical recovery two months later.

In most cases, however, in which anxiety and depression are combined—latently or overtly—recovery from the mental-emotional disturbance as well as recovery from the disturbance of the autonomic nervous system can be brought about by properly timed and spaced shift from nonconvulsive to convulsive electric treatment.

An illustrative case is that of a 45 year old woman who consulted me in a state of severe agitated depression in which tension, bewilderment, depersonalization, and confusion of thinking were prominent features of the symptomatology. She did express depressive self-reproaches but she was also anxious and fearful, showed blocking, and appeared dazed.

One and one-half years before she had a similar episode for which she had been treated with convulsive electroshock treatment. She had responded well to the first treatment in terms of lessening of depression, but after the second treatment lapsed first into a manic state, then in the course of four more treatments into a state of anxious panic. Because of this alarming build-up of anxiety, electroshock treatment had to be discontinued after the fifth treatment. The anxious turmoil state in which convulsive electroshock treatments had left her subsided only slowly after four months of hospitalization.

In view of this history I was careful in evaluating her autonomic response pattern prior to making plans for the treatment of her present similar episode. The pretreatment Funkenstein test (Fig. 54 A) showed that there was a moderate reaction to epinephrine but with precipitation of severe anxiety followed by a chill during which the blood pressure could not be obtained because of the noise engendered by the muscles. There was a slight reaction to mecholyl with a chilly sensation after the blood pressure had returned to the preinjection level. At the start of the mecholyl reaction there was some anxiety precipitated. Clinically, during the administration of the test, the patient appeared not only depressed but extremely fearful to such a degree that it constituted almost a panic. Dr. Funkenstein, who saw the patient in consultation with me, agreed that in previous experience with patients with this type of autonomic reaction they usually did well on a few shock treatments but were apt to become worse if the series was continued as the anxiety precipitated by epinephrine became more severe. Treatment which would decrease this factor was expected to be of aid in this patient. Nonconvulsive treatment was decided on as the treatment to start with.

A day after the first nonconvulsive treatment the patient stated, "I feel better, clearer, less confused." She appeared at ease, was much more animated and alert, and she found that she was able to read a bit for the first time since the onset of her illness. Ability to sleep at night had been restored after the first treatment. After four nonconvulsive treatments the patient stated that her mind was clear, that she was no longer confused or fearful, but that she was beginning to be more depressed; she had a feeling that she wanted to let loose and cry so that she could get back and be friends with herself. As soon as lachrymose depression thus came to the fore, combined convulsive-nonconvulsive treatment was instituted. During the next ten days four combined convulsive-nonconvulsive treatments were given while at the same time some of the dynamic sources of her self-driving attitudes, including her introjected mother image, were worked through psychotherapeutically. After a total of four nonconvulsive treatments and four combined convulsive-nonconvulsive treatments had been administered, she and her husband stated that she was in every way her own best self except for slight "lack of pep." The basal autonomic nervous system was retested at this time (Fig. 54 B). The basal blood pressure was lower than on the previous test. There was a marked rise in blood pressure after epinephrine with the precipitation of a mild anxiety

attack. This was much less severe than previously, and no chill occurred as it had on the previous test. Following mechlolyl there was a minimal reaction without the precipitation of anxiety which had taken place before and again no chill was precipitated. Apart from the very mild anxiety precipitated by epinephrine, the autonomic graph was within normal limits. Soon after her discharge from the hospital 19 days after admission and her first nonconvulsive treatment, the patient achieved full recovery from this particular episode.

In a very similar female patient, aged 43, who also presented a combined picture of severe anxiety and depression, similar tactics of treatment were employed. This case was particularly instructive because the patient had been suffering from a chronic agitated depression for almost twenty years and she was able to give a very clear-cut description of her various subjective feelings. She clearly distinguished a feeling of wretchedness in which she felt like weeping and which predominated during the day, particularly mornings, from her feelings of anxiety with "frightful apprehension, fear of dying and fear of going out of my mind," which belabored her particularly at night when she frequently awoke in a panic. After her first nonconvulsive treatment this patient stated that her feeling of daytime wretchedness had remained unimproved, but for the first time in years she had felt "serene and calm at night. The anxiety, the tossing, the sweat were gone. I didn't really notice that until I woke up in the middle of the night and found that I wasn't in a panic but calm and happy." Her depression was subsequently treated by combined convulsive-nonconvulsive treatment, resulting in significant improvement.

Myoclonic Treatment. The psychophysiologic effects of myoclonic treatment, i.e., of cortical myoclonic stimulation just below the grand mal threshold, are indistinguishable from those of nonconvulsive (vaso-vagal akinetic) treatment. Like the latter it relieves anxiety, increases spontaneity, and restores awareness for memory contents, while it may enhance depression.

Combined Convulsive-Nonconvulsive Treatment. In cases in which depression or other psychotic manifestations implying relative ineffectiveness of defensive operations are as severe as or more severe than the coexisting anxiety, it is best to use combined convulsive-nonconvulsive treatment from the outset. Such combined treatment consists of the administration of nonconvulsive electrostimulation immediately after the convulsive electric treatment. By this means it is possible to avoid completely the rebounding of depression after relief of the anxiety which may occur if nonconvulsive treatment alone is used. In most cases this form of treatment also prevents eruption of epinephrine-precipitable anxiety after relief of depression or other psychotic manifestations, which may occur if convulsive treatment alone is applied.

As far as the reactions of the autonomic nervous system are concerned, combined treatment tends to produce less marked shifts of the basal blood pressure (Figs. 55 and 56) than either convulsive or nonconvulsive treatment alone.

In cases in which only the response to mechlolyl is enhanced (Fig. 55 A), this response becomes normal after treatment (Fig. 55 B) in the same manner in which convulsive treatment alone would correct this type of disturbance. When there is both prolonged enhanced reaction to mechlolyl and anxiety precipitable by epinephrine (Fig. 56 A), combined convulsive-nonconvulsive treatment relieves both the overreaction to mechlolyl and

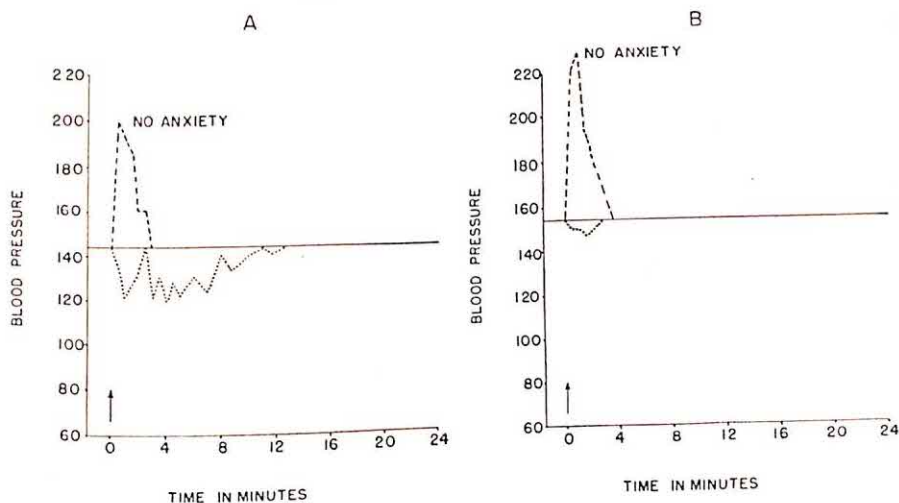


Fig. 55. Male, aged 35, suffering from paranoid psychosis with depressive features of two years' duration, severe of four months' duration.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before treatment. Note slightly enhanced and prolonged fall of blood pressure after injection of mecholyl.

B. Funkenstein test after recovery following two combined convulsive-nonconvulsive treatments of 3 minutes' duration each, administered through temporoparietal leads. Note normal reaction to mecholyl injection.

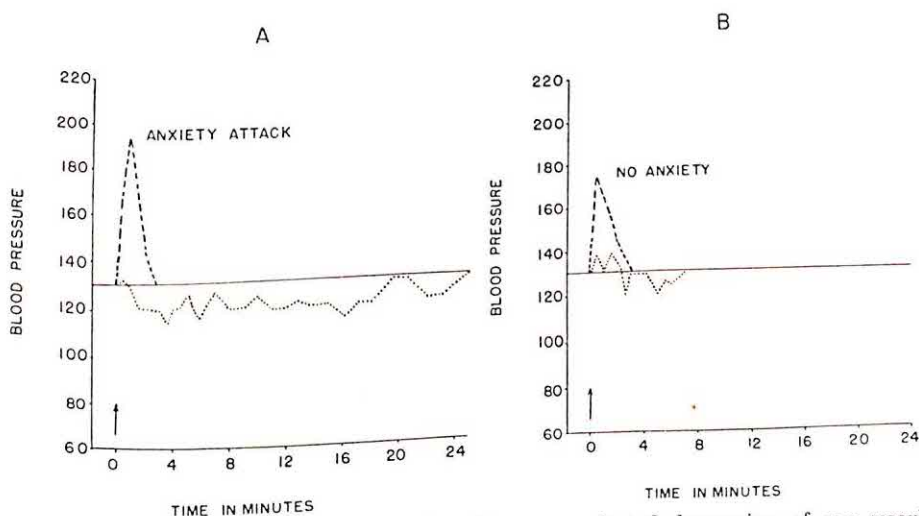


Fig. 56. Female, aged 60 years, suffering from an agitated depression of one year's duration.

The broken line represents the blood pressure response to epinephrine (0.025 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before treatment. Note prolonged fall of blood pressure after injection of mecholyl, and anxiety attack precipitated by epinephrine injection. (Cf. Fig. 51 A.)

B. Funkenstein test repeated after complete clinical recovery following 9 combined convulsive-nonconvulsive treatments of 2 minutes' duration each, administered through frontal leads. Note normal response to mecholyl and absence of epinephrine-precipitable anxiety (cf. Fig. 51 B).

the epinephrine-precipitable anxiety (Fig. 56 B). This effect of combined convulsive-nonconvulsive treatment is similar to that of insulin coma treatment, which also relieves both these disturbances, and in marked contrast to the action of purely convulsive electric shock which tends to aggravate the epinephrine-precipitable anxiety of the type shown in Fig. 56 A. (Compare Figs. 56 A and 56 B which illustrate the response to combined convulsive-nonconvulsive treatment with Figs. 51 A and 51 B which illustrate the response of a similar type of disturbance to convulsive electric shock treatment alone.)

For this reason combined treatment is useful not only for the relief of depressions complicated by anxiety but also for the treatment of schizoaffective, paranoid, and certain schizophrenic states, especially whenever epinephrine-precipitable anxiety is a feature of the autonomic response pattern. The presence of epinephrine-precipitable anxiety causes conventional convulsive electroshock treatment to fail in 63.6 per cent of such cases; while in the overall group of patients free from epinephrine-precipitable anxiety failures with purely convulsive electroshock therapy are reduced to 41.9 per cent; in the subgroup characterized by marked and prolonged overreaction to mechloral in addition to absence of epinephrine-precipitable anxiety, failures are reduced to as little as 9.1 per cent (Funkenstein et al.⁷⁵).

By properly balancing the amount of convulsive and nonconvulsive treatment administered—guided by the clinical psychiatric findings as well as by the autonomic reaction pattern—I was able by means of such combined convulsive-nonconvulsive treatment to reduce the number of failures in an otherwise unselected group of 23 consecutive psychotic patients with epinephrine-precipitable anxiety—comparable to Funkenstein's most unfavorable subgroup—to that prevailing in his most favorable group, namely 9 per cent. After combined convulsive-nonconvulsive treatment, 18 of the 23 patients in whom the Funkenstein test had demonstrated the presence of epinephrine-precipitable anxiety achieved full clinical and/or social recovery (78 per cent), 7 complete (30 per cent), 11 social recovery (48 per cent); 3 additional patients (13 per cent) achieved significant clinical improvement, only 2 cases (9 per cent) being failures. This brings up the total of clinically significantly improved patients in this small series to 91 per cent. Only 12 had marked and prolonged reactions to mechloral favorable for convulsive electroshock treatment; 9 showed slight or moderate overreactions to mechloral; 2 had normal reactions to mechloral (only one of the latter is among the failures; 1 achieved full clinical improvement). In 9 of these cases the series of combined treatments had been preceded by a series of nonconvulsive treatments which had failed to give these patients sufficient relief. In one case the series of combined treatments was followed by two nonconvulsive treatments in order to tone down an anxious aftermath. This patient achieved social recovery at the end of the treatment series, and complete recovery after follow-up psychotherapy. In 13 cases combined treatments were given throughout the entire series from start to finish. The clinical diagnoses in these 23 cases included 15 agitated depressions in a wide range of age groups, varying from 29 to 60 years; 1 case of mania; 3 cases of schizoaffective and 4 of schizophrenic psychoses. The failures of combined treatment were one manic and one schizophrenic, both of whom

had also failed to be sufficiently relieved on preceding purely nonconvulsive electric treatment. Both responded later to insulin treatment.

Combined convulsive-nonconvulsive treatment has additional advantages over purely convulsive electric shock. The incidence and degree of disturbance of awareness for memory contents is greatly reduced; frequently such disturbance is completely eliminated, and there is no reduction in spontaneity. For this reason I have taken to adding a minute of nonconvulsive treatment routinely in most cases which I am treating with convulsive electroshock, even if epinephrine-precipitable anxiety is absent, with the exception of the most severe cases of pure depression, particularly in the involutional group, in whom I wish to avoid anything that could conceivably lower the intensity and irradiation of the supramaximal convulsive stimulus administered by means of alternating current.

With the use of combined convulsive-nonconvulsive treatment it is possible to prevent and to relieve the states of wild, panicky, maniacal, deliriod excitement that sometimes develop as complications caused by purely convulsive electroshock treatment, i.e., the Frosch-Impastato⁷⁰ syndrome. This syndrome never occurred in my large series of patients treated with combined convulsive-nonconvulsive treatments (218 cases, 3087 treatments). In a 42 year old female patient who developed such a syndrome after nine purely convulsive electroshock treatments administered with alternating current—for an entire 24 hour period this patient tried to fling herself against doors and windows, shrieking in fear—one combined convulsive-nonconvulsive treatment instantly and completely relieved it, and seven more combined treatments brought about uneventful recovery from her original paranoid schizophrenic psychotic state.

Combined convulsive-nonconvulsive electric treatment can be administered either through frontal or temporoparietal leads. It seems that its anxiety-relieving effect is somewhat more effective through temporoparietal leads (Alexander²⁸). If longer treatments of more than 7 minutes' duration are used, it is best to administer them through movable hand electrodes. Moving the electrodes forward from the bitemporoparietal position into bifrontal position has an effect similar to that of reducing the current.

In order to balance relief of depression and other regressive psychotic manifestations on the one hand, and anxiety on the other, it is important to proportion the time provided for convulsive stimulation and for nonconvulsive stimulation correspondingly. The autonomic reaction pattern as demonstrated by the Funkenstein test will be an equally important guide. In some agitated depressions, especially in the older age groups, an approximately equal time allowed for the nonconvulsive stimulation (66 seconds) to that occupied by the convulsive phase of the treatment (54 seconds) may be sufficient, making a total treatment time of 2 minutes. This treatment is best given through frontal leads. In case of more severe anxiety, 2 minutes and 6 seconds should be allowed for the nonconvulsive phase of the treatment, thus making a total treatment time of 3 minutes. A treatment of 4 to 6 minutes' duration may be given according to the requirements of the patient. In schizophrenic illnesses in which anxiety is severe the nonconvulsive phase of treatment should be allowed to extend from 7 to 12 minutes. This type of treatment has become known as electric coma or electronarcosis (see Chapter VII). In schizophrenic pa-

tients this type of treatment was found to be only slightly inferior to insulin, but definitely superior to convulsive electroshock (Holt, Landau, and Vernon,⁹¹ Landau and Holt¹⁰³).

Psychophysiological Effects of Insulin Coma Treatment

Insulin coma treatment relieves anxiety, tension, fear, irritability, hostility, elation, paranoid projections, obsessive and compulsive thinking, delusions, hallucinations, profound psychomotor inhibition and other regressive states, as well as depression. It is for this reason that insulin coma treatment is the treatment of choice for schizophrenia. I have found it, however, equally helpful in other chronic emotional disorders such as chronic depressions and chronic manic states which were refractory to electric treatment, as well as in certain obsessive-compulsive states that were refractory to psychotherapy and to combined convulsive-nonconvulsive electric treatment, especially in the younger age groups.

Insulin treatment does not accomplish these results in association with a simple raising of defenses such as occurs after convulsive electroshock treatment; on the contrary, the patient appears divested of his defenses in the early phases of the treatment. On awakening from deep insulin coma, especially if the method of rapid intravenous interruption is used, the fear, panic, suspicion, the feeling of forsakeness and rejection, and the seething hostility of the paranoid schizophrenic are often completely replaced by the trusting confidence and the happy optimism of a cherished child. When the patient is awakened slowly by gastric gavage, there may be a brief flurry of activation of anxiety and fear (see Palisa and Flach²³⁰) at the point when the decorticate phase gives way to partial wakefulness, which becomes, however, rapidly replaced by the characteristic trusting serenity as soon as the patient is fully awakened. This happy, yet insightful defenseless state at first lasts only an hour or two after each treatment; as the day wears on, the old psychotic manifestations recur, but with gradually decreasing severity and persistence (see also Storch²⁶¹). After a number of deep comas, which may vary from ten to thirty, the psychotic manifestations and bristling defenses of patients responding well to treatment recede into the background and gradually vanish, the patient presenting a wide-open, syntonic, defenseless state throughout, eager for communication and psychotherapy. This change in the dynamic constellation continues throughout most of the remainder of the treatment period. As treatments progress further, and during the post-treatment period, the patient gradually becomes able to rebuild his defenses, and to succeed in finding better defensive patterns to build on, especially the very efficient defense of identification, for which insulin coma treatment provides an almost ideal setting. It is obvious that appropriate psychotherapy can be of great help to the patient during this phase, as well as during the partly overlapping preceding phase of lowering of defenses.

Psychosomatic Response Pattern and Clinical Results. The fact that insulin coma treatment relieves anxiety as well as depression, and that it lowers as well as helps reform defenses—which we have come to regard as antipodic on the basis of our experiences with electroshock and electric nonconvulsive stimulation treatment—is reflected also in the changes of autonomic reactivity which accompany successful treatment; that is, it relieves both epinephrine-precipitable anxiety and enhanced responsive-

ness to cholinergic stimuli. Figure 57 A shows the pretreatment Funkenstein test in the case of a young schizophrenic girl showing marked epinephrine-precipitable anxiety as well as a marked and prolonged fall of blood pressure after the injection of mecholyl. A preceding attempt elsewhere to treat her with electroshock was discontinued when her anxiety skyrocketed after the eighth treatment. I repeated the attempt with the same result; the first three treatments alleviated her associated depressive mood disturbance, but after the fourth treatment, her anxiety again

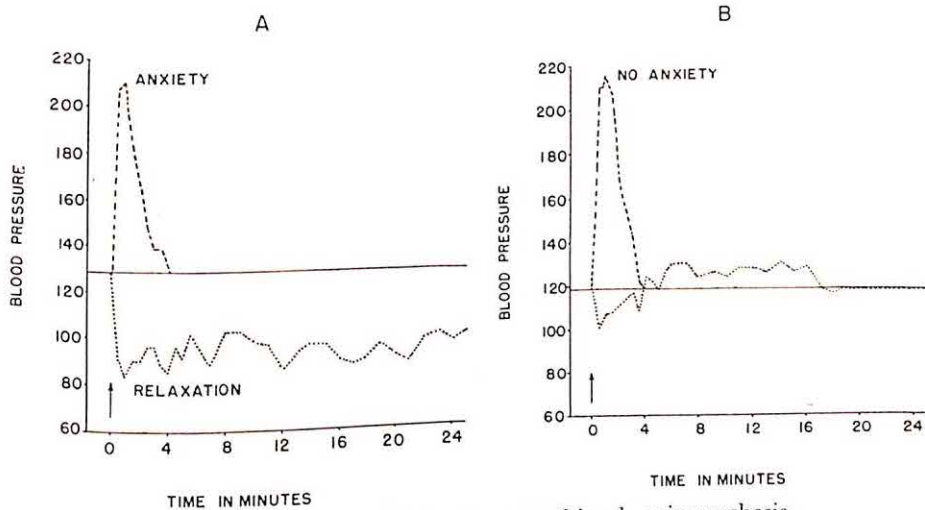


Fig. 57. Female, aged 20 years, suffering from a schizophrenic psychosis. The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).
A. Funkenstein test before treatment. Note marked and prolonged fall of blood pressure after injection of mecholyl, and epinephrine-precipitable anxiety.
B. Funkenstein test after recovery following 62 deep insulin coma treatments. Note absence of epinephrine-precipitable anxiety in spite of slightly enhanced blood pressure response to epinephrine, and normal response to mecholyl injection, apart from slight rebounding overshooting above the preinjection level.

skyrocketed and made further electroshock treatment not only inadvisable, but impossible. After 62 insulin coma treatments, not only did she achieve complete clinical recovery, but in addition, the reactivity of her autonomic nervous system improved significantly (Fig. 57 B). Injection of epinephrine no longer produced anxiety and after an injection of mecholyl her blood pressure dropped only slightly, coming back to the base line within four minutes; the only residual abnormality was that at that point her blood pressure rose slightly above the base line over a period of twelve minutes.

In the case of the 42 year old woman suffering from chronic manic psychosis mentioned previously (p. 232), who had started her treatment series with nine nonconvulsive treatments (see Figures 52 A and B), thirteen convulsive electroshock treatments had left unrelieved both her thymic manic excitation—which soon reverted from a friendly elated pattern back to the previous irritable hostile state—and the associated prolongation of the reaction to mecholyl, although the fall of the blood pressure after injection of mecholyl, while equally prolonged, was not as marked as before. Although epinephrine-precipitable anxiety had not re-

curred, a prolongation of her response to epinephrine (note the second peak in Fig. 58 A)—in spite of simultaneous administration of insulin in subcoma doses—heralded the probable reeruption of epinephrine-precipitable anxiety; hence further continuance of electroshock was considered inadvisable. Deep insulin coma treatment was then instituted. After sixteen comas, the patient achieved complete clinical recovery, and her autonomic reaction pattern returned to normal (Fig. 58 B).

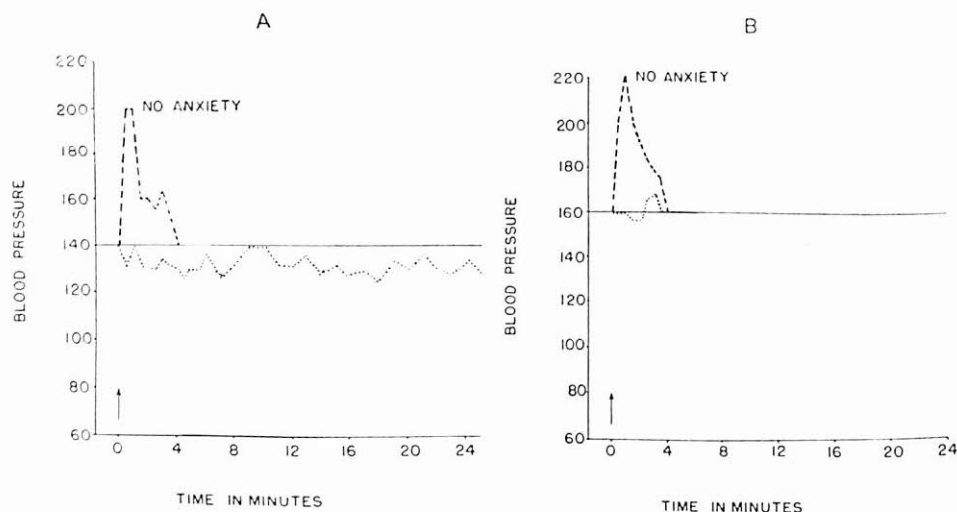


Fig. 58. Female, aged 42 years, suffering from manic psychosis; continued from Fig. 52. The broken line represents the blood pressure response to epinephrine (0.025 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test after 13 convulsive electroshock treatments, the last of them combined with insulin subcoma, administered between July 20 and August 14, 1951. Note that the fall of blood pressure after injection of mecholyl has become less marked, but is still abnormally prolonged. Epinephrine-precipitable anxiety is absent, but blood pressure reaction to epinephrine has become prolonged.

B. Funkenstein test after 21 additional insulin subcoma treatments and 16 deep insulin coma treatments, administered between August 15 and September 28, 1951. At the time of this test the patient had achieved full clinical recovery. Note normal autonomic response pattern, including absence of epinephrine-precipitable anxiety.

Not only does insulin coma reduce excessive reactions to cholinergic as well as adrenergic stimuli, but it is capable of achieving the opposite effect whenever these reactions are inadequate, particularly the reaction to epinephrine. It may thus be stated that insulin actually restores the balance and amplitude of autonomic responsiveness rather than altering the autonomic responses in a specific direction, while the electric forms of treatment are limited to producing only the latter effect. Figure 59 shows the autonomic reaction pattern of a 31-year-old married woman suffering from catatonic schizophrenia with hallucinations. She had experienced a number of previous hallucinatory episodes, the first one at age 15 when she saw God's face, great and gigantic, beaming at her from the sky as she lay in the sun at the beach. A second episode occurred at age 20 from which she recovered spontaneously, but with residuals. The third episode occurred at age 25. At that time, she responded adequately to a series of fourteen electroshock treatments. A fourth episode at 30 remained entirely

unresponsive to thirteen electroshocks, the last treatment being given a week before the autonomic graph shown in Fig. 59 A was obtained. Sixty insulin coma treatments brought about apparent complete recovery which was further consolidated by psychotherapy, which enabled her to assume greater independence from her mother and to be more permissive and less anxious in dealing with her husband and children. Her autonomic graph at the completion of the insulin series (Fig. 59 B) showed that the reaction to epinephrine had become normal while the reaction to mecholyl had become slightly enhanced, still falling, however, into approximately normal limits. Her clinical recovery has been sustained, and her intrapsychic and social maturing continues at a gratifying pace.

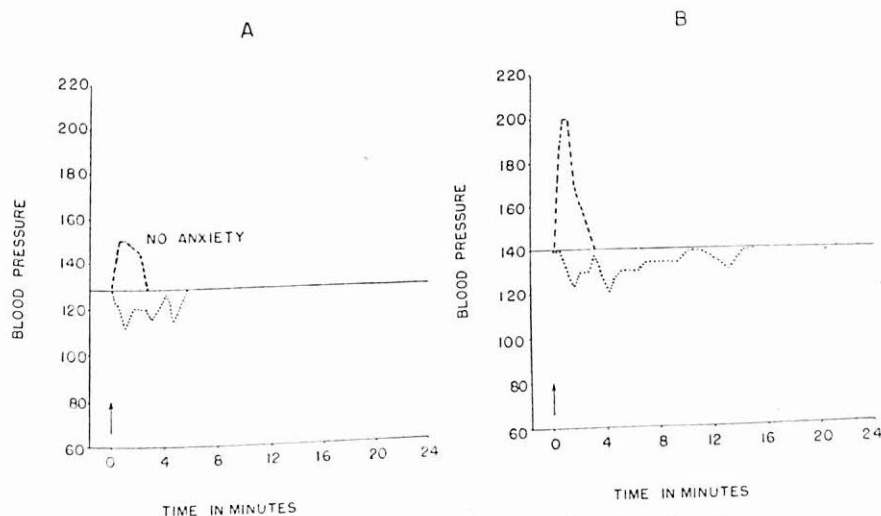


Fig. 59. Female, aged 31 years, suffering from catatonic schizophrenia. The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test before insulin coma treatment. Note inadequate (subnormal) response to epinephrine injection.

B. Funkenstein test after 62 insulin coma treatments. The patient had achieved full clinical recovery at the time of this test. Note restoration of normal response to epinephrine injection as well as the fact that response to mecholyl has also become slightly enhanced and moderately prolonged, still falling, however, into approximately normal limits.

It is interesting that the abolition of epinephrine-precipitable anxiety afforded by insulin coma treatment has a particularly lasting quality. When one schizophrenic patient who achieved clinical recovery after insulin coma treatment (see Fig. 57) suffered a mild relapse in the form of a slight reactive depression with partly somatized anxiety-tension symptoms, the Funkenstein test (Fig. 60) showed that her epinephrine-precipitable anxiety had not recurred, the recurrence being limited to enhanced reactivity to cholinergic stimulation. This was promptly relieved by combined convulsive-nonconvulsive electric treatments followed by psychotherapy which was aided by abreaction treatments under ether given by inhalation. She made an uneventful and prompt recovery and has remained well for the past two and a half years. This case emphasizes that prompt treatment of relapses or threatened relapses are in order, and that

treatment must be adapted not only to the clinical symptoms but also to the nature of the associated disturbance of the autonomic nervous system. It is important to realize that after adequate insulin coma treatment, such relapses may be only partial and mild and do not necessarily require full retreatment by insulin coma. This case also emphasizes the fact that as defenses are strengthened after treatment, new stress may produce a less severe breakdown, in this case, a mild reactive depression, or a neurotic-depressive breakdown rather than a schizophrenic breakdown. Conversely, an untreated mild neurotic breakdown may leave the personality open to the development of a more severe, namely, schizophrenic breakdown when subjected to recurrent or more severe stress. This clearly

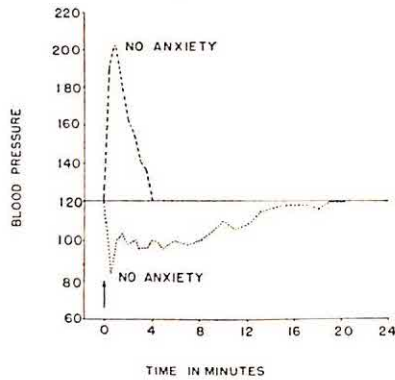


Fig. 60. Female, aged 21 years, suffering from a mild relapse in the form of depression and "neurotic" symptoms after recovery a year before from a schizophrenic psychosis following 62 insulin coma treatments. The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly). (Previous Funkenstein graphs for this same patient are shown in Fig. 57.) Note the fact that epinephrine-precipitable anxiety has not recurred, and that the fall of her blood pressure after injection of mecholyl was neither as marked nor as prolonged as during her previous psychotic episode. (Cf. Fig. 57 A.)

indicates that the various clinical syndromes may represent receding or advancing movements in the defensive position of the patient rather than different nosologic entities.

It is very difficult to establish a clear-cut expectancy rate of recovery on the basis of the data in the literature, since the quality of treatment employed was obviously quite heterogeneous. Sakel's^{250, 251} original idea of easing the patients gradually into and out of coma included four phases: preparatory dosage, severe hypoglycemic shocks, rest days, and terminal or polarization phase. He advised intravenous termination only in emergencies, believing that gradual awakening by tube with its accompanying transitional stages was equally as important for the success of treatment as the gradual preparation for coma with progressive doses of insulin.

In the light of the Funkenstein test and our experience with the way in which defenses shift in accordance with the autonomic reaction pattern, we can see now what this method accomplishes: namely, a balanced stimulation both of the cholinergic system and the adrenergic system, the latter particularly in evidence during the phase of rage reaction at the end of the decorticate phase of coma. Both parts of the autonomic nervous system receive strong stimulation during insulin coma treatment—of that

there can be no doubt. An interesting correlated observation indicating marked stimulation of the adrenergic system is the early observation by Gross,¹⁹³ who found that the eosinophils decline during coma, and that this decline outlasts the coma itself. Kersley and his coworkers²⁰⁸ found a significant fall in circulating eosinophils as well as marked improvement of rheumatoid arthritis as results of insulin coma treatment, while no comparable significant effects were observed in a smaller series of such patients treated by electric shock. Shands and Menzer²⁵⁷ found that of those schizophrenic patients in whom the eosinophils rose instead of falling, 75 per cent were therapeutic failures, while in the group in whom eosinophils declined, 67 per cent were classed as successes with therapy. Unfortunately, Shands and Menzer do not give any data indicating at what precise phase of decortication and decerebration respectively treatment was stopped. It is possible that some of their treatments were terminated at the sympathomimetic phase, causing the eosinophils to drop, or at the parasympathomimetic phase, causing the eosinophils to rise. There may be a further correlation of this fact clinically; in my personal observation of a large number of cases, I have found that the phase of rage reaction is almost always a stormy one in paranoid and catatonic patients, while this phase seems to be by-passed in cases of simple schizophrenia, hebephrenia, and in most patients with obsessive-compulsive states. Moreover, it is established that recovery rates are greater in the paranoid and catatonic groups than in other forms of schizophrenia, especially the hebephrenic group. Further studies of these relationships will supply us with more adequate data for the indication of the specific degrees and phases of coma to be used in specific conditions.

It can readily be seen that insulin therapy—even insulin coma treatments—may be physiologically quite different things in the hands of different therapists. In our gradual acquisition of knowledge of the specific rationale for the specific disorder, Sakel's idea that successful insulin coma treatment must include a significant amount of release of activity at all levels seems to be borne out, thus explaining the improved function in both sympathetic and parasympathetic levels of the autonomic nervous system observable in all successfully treated cases. It is definite that when treatment is limited in terms of inadequate numbers of treatments (such as a small series of 20 treatments) or in terms of inadequate depth of coma, the results are less good (45 per cent total improved, including 30 per cent complete and/or social recovery) (Gottlieb and Huston,¹⁹⁰ Zijlstra²⁷⁶), which is less than the results obtainable with the standard Sakel method, to be discussed further, below.

On the other hand, if the subcoma phase is entirely avoided and the patients thrown precipitously into coma by means of intravenous injection of insulin, results are also less good (41.9 per cent total improved, 29 per cent clinical and/or social recovery)* than with the standard method.

Until we know more surely the precise phase of coma treatment needed for the relief of a specific type of mental disorder, a complete coma treatment which goes through the entire range of the phases is best. This of course requires personnel well trained in the principles involved so that they can apply the treatment with courage, determination, and skill; therapists secure in the belief that they can treat emergencies when they

* See refs. 199, 205, 215, 233, 234, 239, 252, 258.

arise, or better still, forestall them, without the fear of treatment that causes those therapists insufficiently well versed in the method to withhold adequate depth of treatment from their patients.

Statistics of Treatment Results. Comparison of results between the grand totals of schizophrenics treated with insulin coma according to the standard Sakel method,* and schizophrenics both from the preinsulin era and from certain control studies of more recent date† present an impressive difference; clinical improvement was achieved by only 29.3 per cent of the control group numbering 11,080 patients (Table 11), but by 61.3 per cent of the insulin coma-treated group, numbering 9483 patients (Table 18). Further investigation reveals a sharp contrast between 18.9 per cent complete and/or social remissions in the control group (Table 11) and 47.6 per cent parallel remissions in the group treated with insulin coma (Table 18). The figures for complete recovery in the control group are 5 per cent (Table 12), while they comprised 21 per cent of the total insulin coma-treated group (Table 19). It is, of course, important to bear in mind, as Malzberg²¹⁷ pointed out, that the insulin-treated group represents a selected group from the point of view of physical factors, whereas many of the controls were relegated to the untreated group because their physical condition contraindicated insulin therapy. On the other hand, a good many patients in the insulin-treated group, particularly in the first years following initiation of insulin coma treatment, were "hopeless" chronics and deteriorated schizophrenics for whom insulin coma treatment was attempted as a last resort.

It is an unfortunate fact that any statistical analysis of the above groups is faced with the problem of coping with each therapist's individual—and in many cases, unique—method of presenting his results. There would appear to be a great need for standardization of presentation in this area. Since it is generally agreed that duration of illness before the initiation of treatment has a good deal of bearing on the results obtainable, it is practically meaningless to publish a series of results without including this data; it is equally important to present the diagnostic subclassification of schizophrenia together with their individual durations of illness, if any parallel conclusions as to the varying degrees of response of these subclassifications to insulin treatment are to be drawn. In this respect, much of the data is, regrettably, incomplete. However, utilizing the data available, it can be seen that the highest rate of improvement—97.2 per cent—is obtained in the catatonic group with a duration of illness of from one to six months before treatment was initiated, 94.8 per cent in the parallel paranoid group, and 86.6 per cent in the hebephrenic group. Grading downward in chronicity to the two to five year group, we find 51.2 per cent total improvement in the paranoid group, 30.8 per cent in the catatonic, and 46.0 per cent in the hebephrenic (Table 18). There is a consistent further down-grading of results correlated with duration of illness prior to treatment in all three categories of improvement, namely: total sig-

* See refs. 159, 160, 161, 162, 163, 164, 166, 167, 168, 169, 171, 173, 174, 175, 176, 178, 179, 180, 182, 183, 184, 186, 187, 188, 189, 194, 195, 196, 197, 198, 203, 204, 206, 207, 210, 211, 212, 213, 214, 217, 218, 219, 220, 221, 222, 223, 224, 226, 227, 229, 231, 232, 235, 236, 237, 238, 240, 241, 243, 244, 246, 247, 248, 249, 254, 255, 256, 259, 262, 263, 264, 267, 268, 269, 270, 271, 273, 274, 275.

† See refs. 164, 169, 171, 194, 202, 212, 213, 214, 217, 223, 227, 228, 232, 236, 242, 243, 246, 254, 259, 260, 272.

nificant clinical improvement, complete and/or social recovery (Table 18), and complete recovery (Table 19). The figures for significant clinical improvement, for instance, decrease from a high of 90.2 per cent for patients ill one to six months, to a low of 46.3 per cent for patients ill five to ten years.

A comparative survey of the control figures indicates an improvement rate of 48.5 per cent in the group classified as "acute" (duration of illness from one to eighteen months) and 27.7 per cent in the "chronic" group (duration of illness over eighteen months) (Table 11). When subclassification breakdowns are available but the duration of illness is unknown, the control group of paranoid schizophrenics achieved 11.9 per cent complete and/or social remission, and 23 per cent total significant clinical improvement (Table 11), as compared with 31.5 per cent full and/or social recovery, and 52.6 per cent total significant clinical improvement (Table 18) in the parallel group of insulin-treated paranoids; control catatonics achieved 30.4 per cent complete and/or social remissions and 37.9 per cent total improvement (Table 11), as contrasted with 37.8 per cent complete and/or social remissions and 59.4 per cent total significant clinical improvement in the group of catatonics of unknown duration treated with insulin (Table 18). Hebephrenics achieved the least favorable percentages, namely, 15.1 per cent and 27.0 per cent respectively in the control group (Table 11), as against 20.9 per cent and 42.5 per cent in the insulin-treated group (Table 18).

Of a group of 147 patients suffering from schizodepressive and other schizoaffective states, 48.3 per cent achieved complete and/or social remissions, and 59.8 per cent total significant clinical improvement (Table 20).

Although insulin coma therapy is predominantly used for the treatment of schizophrenic and closely related psychoses, it is important to realize that it is highly effective in other mental-emotional disorders as well. As tabulated in Table 20, 88 manic-depressives, manic phase, attained a 90.9 per cent significant clinical improvement rate, and an equally impressive 89.7 per cent rate of complete and/or social remission. Seventy-one patients suffering from a depressed phase of manic-depressive psychosis achieved 83 per cent total significant clinical improvement, while 74.7 per cent gained complete and/or social remissions. Of 10 manic-depressives, mixed phase, 7 were treated successfully, achieving complete and/or social remissions; and of 34 patients with involutional melancholia, 26, or 76.4 per cent, gained complete or social remissions. I can confirm the fact that insulin coma treatment, while more cumbersome, is highly effective in the affective psychoses, in the chronic type frequently more so than convulsive electroshock treatment. The reason for this not generally appreciated fact is probably that the action of insulin upon the autonomic nervous system is less unilateral than that of convulsive electroshock, as pointed out above. This may be the reason why it was found to be also highly effective in the treatment of a small group of severe psychoneuroses (Table 20), to which I can add a small number of successfully treated chronic anxiety neuroses and compulsive-obsessive neuroses of my own experience, including a recent particularly outstanding treatment result consisting in complete recovery from a severe compulsive-obsessive neurosis that had resisted all other forms of therapy save lobotomy.

Insulin coma treatment, like electroshock, when it is successful, also greatly reduces the duration of hospitalization. One of the most material benefits of insulin coma therapy is the resultant shortening of the hospitalization period for patients so treated in comparison with similar groups of patients hospitalized under custodial and supportive care. Shurley and Bond's²⁵⁹ report of the results tabulated by the Temporary Commission on State Hospital Problems of New York compares 1128 insulin-treated schizophrenics with 876 similar patients who received merely supportive treatment. Of the insulin treated group 79.5 per cent were able to leave the hospital compared with 58.8 per cent of the untreated group. The hospitalization period was appreciably shortened for the insulin group by an average of 3.8 months. This survey estimates that insulin coma therapy carried out in 1128 patients saved 29,695 days of hospital care, and \$80,274 in maintenance costs.

Similarly, Finiefs¹⁸⁸ found that in comparing 446 untreated cases with 563 insulin-treated patients, the treated group averaged 5 months of hospitalization as against 8.3 months for the controls, a saving of 3.3 months per patient, which is in agreement with the New York survey above. Taylor's²⁶³ study of 214 treated schizophrenics concluded that insulin coma treatment saved the state of New Jersey 2409 hospitalization weeks and \$24,000 in costs. Bond's continued follow-up study in 1941¹⁶⁹ presented for contrast the fact that 95 per cent of those patients who recovered with insulin coma treatment did so promptly, attaining discharge or parole within a month of the treatments, whereas those control patients who recovered with custodial care required one to three years to attain this improved status.

Ross, Rossman, Cline, Schwoerer and Malzberg,²⁴⁶ in an analysis of 1039 insulin-treated patients, found that 37.3 per cent were living in the community two years after the completion of treatment, while of 10,140 schizophrenics hospitalized between 1909 and 1911, only 23.3 per cent were living in the community two years after their initial hospitalization. Moreover, of those patients insulin-treated within six months of the onset of their illness, 60 per cent were living in the community two years after hospitalization.

There is no doubt that insulin coma treatment is relatively the most effective treatment of schizophrenia. As Bennett¹⁶⁵ stated, it surpasses other therapies in speed, frequency, durability, and quality of improvement attainable. Our present overall statistical study which lends significant support to this statement is based on a total of 29,383 cases (Table 21). The clear-cut conclusion emerges from this large case material that insulin coma treatment is superior to any other form of treatment of schizophrenia (Tables 18, 19, and 21), including custodial and supportive treatment (Tables 11 and 12); intensive psychotherapy (Tables 7 and 8); and convulsive shock therapy (Tables 15 and 16).

The total significant improvement rate of schizophrenic patients under conditions of custodial and supportive treatment is 29.3 per cent of 11,080 cases; with intensive psychotherapy, 38.2 per cent of 1463 cases; with convulsive shock therapy, 49.1 per cent of 7357 cases; while with insulin coma therapy it is 61.3 per cent of 9483 cases (Table 21).

Quality of Improvement in Schizophrenia. Rates for complete and/or social remission in schizophrenia are 18.9 per cent in the group given

custodial and supportive treatment, 18.3 per cent for the group treated with intensive psychotherapy. These two figures do not differ significantly from each other. The rates for complete and/or social remission, however, are 29.1 per cent for the convulsive shock-treated group, and 47.6 per cent for the group treated with insulin coma therapy (Table 21). These two rates are significantly different—below the 0.01 level—from each other and from the rate obtainable by custodial, supportive and/or intensive psychotherapy; i.e., there is less than one chance in one hundred that these differences may have resulted from chance sampling alone. Only 5 per cent of the schizophrenic patients treated with custodial and supportive care achieved complete recovery; 10.6 per cent of those treated with intensive psychotherapy recovered fully, 16 per cent of the group treated with convulsive therapy and 21 per cent of the group treated with insulin coma therapy attained complete recovery (Table 21, Fig. 61 A). These figures are significantly different from each other below the 0.01 level. These figures are capable of throwing some light on the relative quality of improvement following these various types of treatment, a subject of particular interest since Sargant and Slater²⁵³ first suspected that the quality of improvement obtained by insulin coma therapy may be superior. They stated that there is “a less unanimous impression that the quality of remission obtained with insulin is better than that of the spontaneous remission. This is likely to be true, if for no other reason than that recovery takes place earlier under treatment and there is less time for the psychological scarring that is the most terrible effect of the disease.”

Our cumulative figures present actual evidence for a better quality of improvement with intensive treatment, insulin coma or otherwise, as well as with insulin coma treatment in particular. The patients achieving complete remissions comprise 32.0 per cent of the total improved patients in the insulin coma-treated group; those in the convulsive shock treated group and in the group treated with intensive psychotherapy comprise only slightly less, namely, 27.5 per cent and 29.6 per cent respectively, while of those patients who improved under custodial and supportive care, only 18.2 per cent of the total improved group achieved complete remission (Table 22, Fig. 61 B). The superiority of insulin is more strikingly evident if the rates for complete and/or social recovery are taken as the basis for comparison. These are 77.6 per cent of the total improved group for insulin coma therapy, while those for the other three forms of treatment range from 47.9 per cent to 64.2 per cent (Table 22).

Prognostic Criteria in Schizophrenia. In a study of prognostic criteria in schizophrenia, Cheney and Clow¹⁷⁸ concluded that the following composite picture offers the best outlook for remission following insulin coma therapy: a male patient, under 30 years of age, who had an adequate prepsychotic personality, whose psychosis was abrupt in onset with a definite exogenous precipitating factor, whose illness was of less than one year's duration before treatment was instituted, and whose illness takes the form of excited catatonia without evidence of deterioration. Conversely, these authors found no direct correlation between improvement and the amount of dosage of insulin, the number of convulsions, or number of comas experienced. The patient's attitude toward treatment had no significant bearing on the final result.

On the other hand, Gralnick^{191, 192} reported slightly better results with

Table 18. Insulin Coma Treatment of Schizophrenia.
Complete and/or Social Recovery, Total Significant Clinical Improvement

TYPE	TOTAL PTS. ILL	DURATION OF ILLNESS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
			CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.	
Paranoid	58	1-6 mos.	43	11	54	74.1	94.8	79.7
	32	6 mos.-1 yr.	17	11	28	53.1	87.5	60.7
	49	1-2 yrs.	28	12	40	57.3	81.7	70.0
	133	2-5 yrs.	46	22	68	34.5	51.2	67.6
	3	5-10 yrs.	2	0	2			
	1	10 plus yrs.	0	1	1			
	1034	Dura. not stated	326	219	545	31.5	52.6	59.9
Total	1310		462	276	738	35.2	56.3	62.6
Catatonic	70	1-6 mos.	51	17	68	72.8	97.2	75.0
	18	6 mos.-1 yr.	9	6	15	50.0	83.3	60.0
	49	1-2 yrs.	12	22	34	24.5	69.5	35.3
	52	2-5 yrs.	10	6	16	19.2	30.8	62.5
	9	5-10 yrs.	0	2	2			
	1	10 plus yrs.	0	0	0			
	1041	Dura. not stated	394	224	618	37.8	59.4	63.9
Total	1240		476	277	753	33.5	60.7	63.3
Hebephrenic	60	1-6 mos.	45	7	52	75.0	86.6	86.6
	23	6 mos.-1 yr.	12	7	19	52.1	82.6	63.2
	28	1-2 yrs.	17	2	19	60.7	67.1	89.5
	115	2-5 yrs.	45	9	54	39.1	46.0	83.2
	4	5-10 yrs.	1	0	1			
	1	10 plus yrs.	0	0	0			
	684	Dura. not stated	143	148	291	20.9	42.5	49.2
Total	915		263	173	436	28.8	47.6	60.4
Simplex	3	1-6 mos.	3	0	3			
	0	6 mos.-1 yr.	0	0	0			
	1	1-2 yrs.	1	0	1			
	6	2-5 yrs.	2	2	4			
	0	5-10 yrs.	0	0	0			
	0	10 plus yrs.	0	0	0			
	49	Dura. not stated	13	14	27	26.5	55.1	48.2
Total	59		19	16	35	32.2	59.4	54.3
Unclassified Schizophrenia	144	1-6 mos.	97	28	125	67.3	86.8	77.6
	326	6 mos.-1 yr.	192	48	240	58.8	73.6	80.0
	138	1-2 yrs.	67	46	113	41.3	81.8	59.3
	275	2-5 yrs.	97	96	193	35.2	70.1	50.3
	131	5-10 yrs.	28	35	63	21.3	40.4	44.5
	31	10 plus yrs.	6	15	21	19.3	67.7	28.6
	4914	Dura. not stated	2808	280	3088	57.0	62.4	90.9
Total	5959		3295	548	3843	55.3	64.6	85.7
Totals	335	1-6 mos.	239	63	302	71.3	90.2	79.3
	399	6 mos.-1 yr.	230	72	302	67.9	75.6	76.2
	265	1-2 yrs.	125	82	207	47.2	78.2	60.4
	581	2-5 yrs.	200	135	335	34.4	57.5	59.7
	147	5-10 yrs.	31	37	68	21.1	46.3	45.6
	34	10 plus yrs.	6	16	22	17.7	64.7	27.3
	7722	Dura. not stated	3684	885	4569	47.8	59.1	80.7
TOTAL	9483		4515	1290	5805	47.6	61.3	77.6

Table 19. Insulin Coma Treatment of Schizophrenia. Complete Recovery

TYPE	TOTAL PTS. ILL	DURATION OF ILLNESS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE	
			CR	SR plus IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL	OF TOT. SIG. CLIN. IMP.
Paranoid	16	1-6 mos.	10	4	14	62.5	71.4
	13	6 mos.-1 yr.	8	5	13	61.6	61.6
	11	1-2 yrs.	3	6	9	27.3	33.3
	28	2-5 yrs.	3	17	20	10.7	15.0
	0	5-10 yrs.	0	0	0		
	0	10 plus yrs.	0	0	0		
	557	Dura. not stated	71	286	357	12.7	19.9
Total	625		95	318	413	15.2	23.0
Catatonic	22	1-6 mos.	20	2	22	90.9	90.9
	10	6 mos.-1 yr.	4	4	8	40.0	50.0
	11	1-2 yrs.	3	5	8	27.3	37.5
	15	2-5 yrs.	2	5	7	13.3	28.6
	4	5-10 yrs.	0	1	1		
	0	10 plus yrs.	0	0	0		
	545	Dura. not stated	85	254	339	15.6	25.1
Total	607		114	271	385	18.8	29.6
Hebephrenic	14	1-6 mos.	10	2	12	71.5	83.3
	12	6 mos.-1 yr.	6	5	11	50.0	54.5
	8	1-2 yrs.	4	4	8	50.0	50.0
	5	2-5 yrs.	1	3	4		
	4	5-10 yrs.	0	1	1		
	0	10 plus yrs.	0	0	0		
	331	Dura. not stated	16	147	163	4.8	9.8
Total	374		37	162	199	9.9	18.6
Simplex	2	1-6 mos.	1	1	2		
	0	6 mos.-1 yr.	0	0	0		
	1	1-2 yrs.	0	1	1		
	6	2-5 yrs.	1	3	4		
	0	5-10 yrs.	0	0	0		
	0	10 plus yrs.	0	0	0		
	27	Dura. not stated	1	19	20	3.7	5.0
Total	36		3	24	27	8.3	11.1
Unclassified Schizophrenia	202	1-6 mos.	107	61	168	53.0	63.7
	183	6 mos.-1 yr.	79	60	139	43.2	56.8
	195	1-2 yrs.	48	111	159	24.6	30.2
	232	2-5 yrs.	30	109	139	12.9	21.6
	109	5-10 yrs.	7	55	62	6.4	11.3
	33	10 plus yrs.	5	17	22	15.1	22.7
	1045	Dura. not stated	240	435	675	23.0	35.6
Total	1999		516	848	1364	26.9	37.8
Totals	256	1-6 mos.	148	70	218	57.8	67.8
	218	6 mos.-1 yr.	97	74	171	44.5	56.7
	226	1-2 yrs.	58	127	185	25.7	31.3
	286	2-5 yrs.	37	137	174	12.9	21.3
	117	5-10 yrs.	7	57	64	6.0	10.9
	33	10 plus yrs.	5	17	22	15.2	22.7
	2505	Dura. not stated	413	1141	1554	16.5	26.6
TOTAL	3641		765	1623	2388	21.0	32.0

female patients than with males; Tennent²⁶⁵ found that prognosis with insulin coma treatment was superior in acute paranoia to acute catatonia, although he found that this situation reversed itself with the increasing

Table 20. Insulin Coma Treatment of Other Psychoses

TOTAL NO. PTS.	DIAGNOSIS	NUMBER OF PATIENTS RESPONDING TO TREATMENT			PERCENTAGE		
		CR plus SR	IMP.	Total No. Sig. Clin. Imp.	OF TOTAL NO. ILL		OF TOT. SIG CLIN. IMP.
					CR plus SR	Total Sig. Clin. Imp.	CR plus SR
147	Schizo-affective de- pressions	71	17	88	48.3	59.8	80.6
14	Psychopathic states	1	3	4	7.1	28.5	25.0
88	Manic-depressive manic	79	1	80	89.7	90.9	98.9
71	Manic-depressive depressed	53	6	59	74.7	83.0	89.9
10	Manic-depressive mixed	7	0	7	70.0	70.0	100.0
34	Involuntal melan- cholia	26	0	26	76.4	76.4	100.0
131	Undiag. psychoses	64	18	82	48.0	62.6	78.1
12	Psychoneuroses, incl. comp.-obs.	8	2	10	66.7	83.3	80.0
Total	507	309	47	356	61.0	70.3	86.8

Table 21. Comparison of Results of Treatment of Schizophrenia by Various Methods

MEANS OF TREATMENT	TOTAL SIG. CLIN. IMP. %	COMPLETE AND/OR SOCIAL RECOVERY %	COMPLETE RECOVERY %
Custodial and Supportive Treatment 11,080 Cases	29.3	18.9	5
Intensive Psychotherapy 1,463 Cases	38.2	18.3	10.6
Convulsive Shock Therapy 7,357 Cases	49.1	29.1	16
Insulin Coma Treatment 9,483 Cases	61.3	47.6	21
Total 29,383 Cases			

chronicity of these two forms of schizophrenia. Taylor,²⁶³ however, achieved least good results in paranoid states and found these patients to have not only the least good prognosis, but also the highest percentage of

relapses; whereas paranoids comprised only 28 per cent of his total group of 214 patients, they constituted 50 per cent of the returnees.

Cohen¹⁸¹ found that those patients who make a decided weight gain with insulin treatment (over 30 pounds) will do approximately twice as well as those who fail to gain significantly, although, of course, therapeutic failure may occur in spite of gross weight gains.

Abnormalities in cerebral electroactivity prior to treatment are considered as connoting unfavorable prognosis (Turner et al.²⁶⁶).

Relapses after Insulin Coma Treatment for Schizophrenia and Their Management. Individual reports of relapse rates of schizophrenic patients treated with insulin coma vary markedly. Horwitz et al.²⁰⁰ summarized available relapse figures from the literature and found that of a total of 303 patients who had attained complete or social remissions, 10.5 per cent eventually relapsed, and of this number, 16 per cent again achieved remissions. Bond, Hughes and Flaherty¹⁷⁰ reported that the original relapse rate of 18 per cent of their 82 patients rose within two years to 30 per cent; Liberson²¹² found that one to three years after termination of treatment, the total clinical improvement rate had fallen from 62 per cent to 40 per cent. Palmer et al.²³¹ found that 8 per cent of their 393 patients relapsed;

Table 22. Treatment Results in Schizophrenia.
The Two Top Grades of Improvement in Per Cent of Improved Cases

	COMPLETE AND/OR SOCIAL RECOVERY %	COMPLETE RECOVERY %
Custodial and Supportive Treatment	64.2	18.2
Intensive Psychotherapy	47.9	29.6
Convulsive Shock Therapy	59.2	27.5
Insulin Coma	77.6	32

McKendree²²⁰ reported a 16 per cent relapse rate from eighteen months to four years after the termination of treatment; Malzberg,²¹⁷ 24.5 per cent within one year, Bond and Rivers,¹⁷¹ 21 per cent within seven years. Gralnick¹⁹¹ found that of 554 patients in his first survey, 50 per cent were paroled at some time, but that the eventual total relapse rate rose at first sharply from 20 per cent at the end of the first year to 38 per cent by the end of the second year, and 46 per cent at the end of the third year, but then only slightly, namely to a total of 47 per cent at the end of four to five years, levelling off at this point, indicating that relapse is unlikely once recovery has lasted four years. Bond and Rivers¹⁷¹ reported a not dissimilar levelling off at a total of about 33 per cent in the second, third and fourth years. However, Gralnick's later study¹⁹² of a smaller group of schizophrenics—158 patients—attained better results. Only 20 per cent relapsed within two years, instead of the anticipated 30 to 40 per cent, a difference which he attributed to the application of more stringent standards for parole. Shurley and Bond²⁵⁹ found that two-thirds of all relapses

occurred within one year after termination of treatment, and that thereafter the relapse rate decreased progressively.

Rupp and Fletcher²⁴⁸ found that in their four-year follow-up study of 129 patients, there were no irreversible relapses in the group which had attained complete or social remission with insulin coma treatment (17.5

PERCENTAGE OF ALL PATIENTS

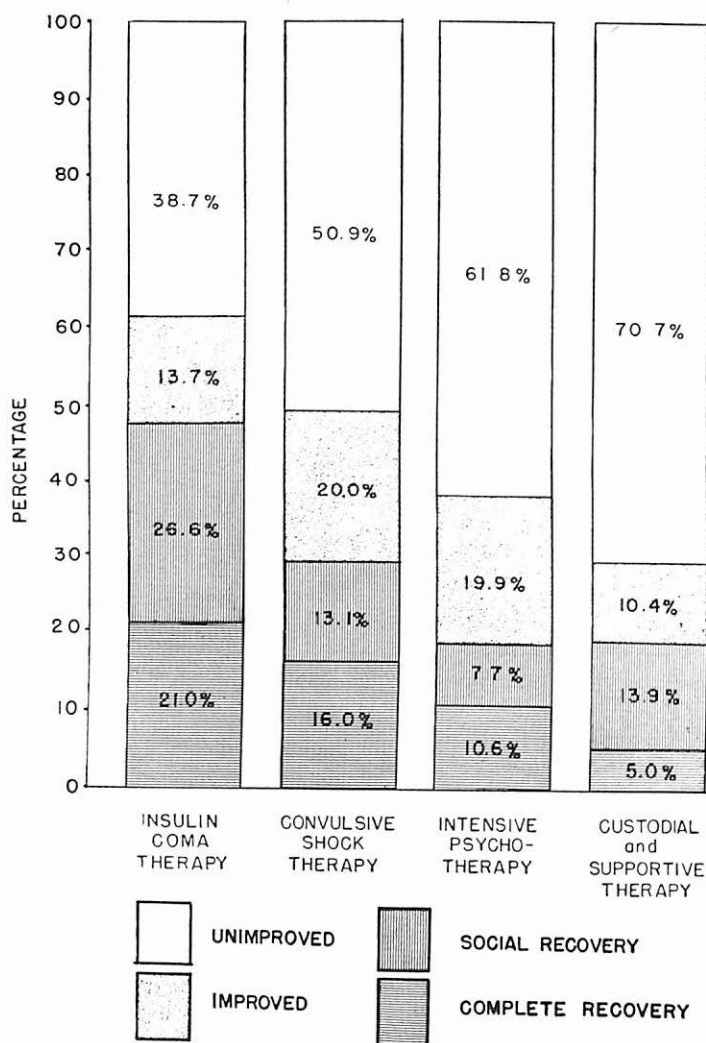


Fig. 61. A. Treatment results in schizophrenia. Tabulation of 29,383 cases. The varying degrees of improvement and the unimproved group in percentage of total patients treated.

per cent relapsed in this group, but all re-recovered with further treatment). Ten per cent of the parolees in the merely improved group relapsed; apparently permanently. These findings indicate that the quality of improvement is an important factor in its stability. Gralnick emphasized that 66 per cent of those who maintained their recoveries had been ill less

than one year before treatment was instituted. However, Horwitz et al.²⁰⁰ in a survey of available relapse figures concluded that there was no correlation between relapse rate and previous duration of illness, nor could they find evidence to support the theory that those patients who achieve less complete remissions are more susceptible to relapse. On the other hand,

PERCENTAGE OF IMPROVED CASES

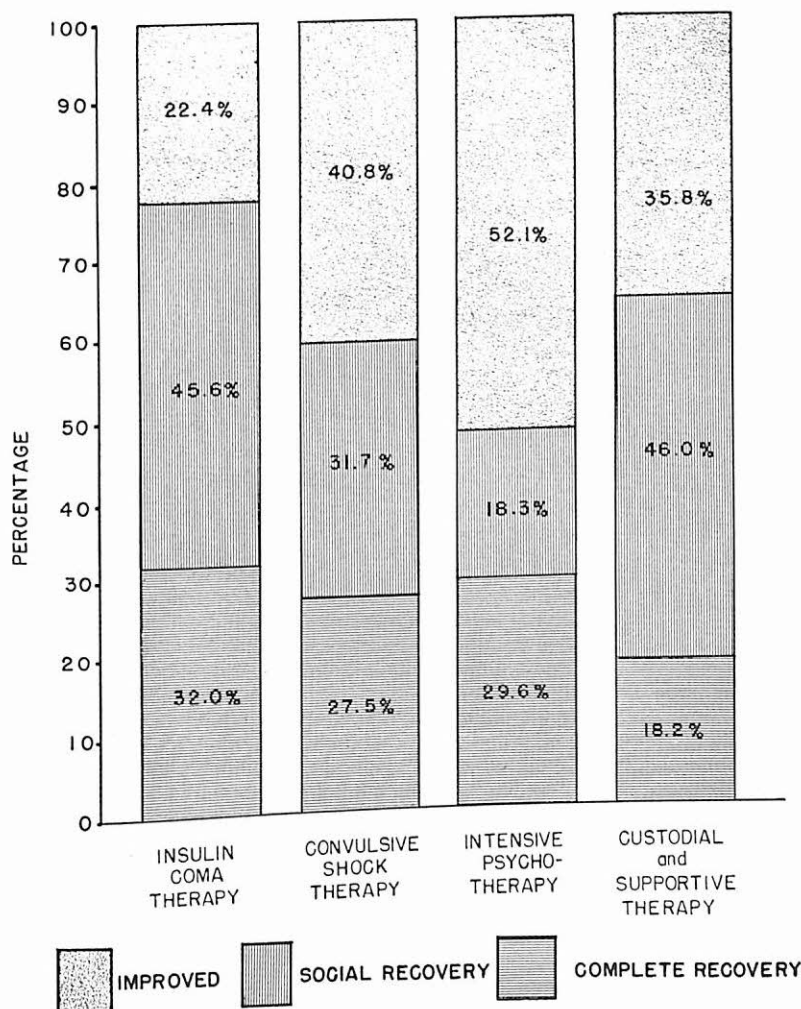


Fig. 61. B. Treatment results in schizophrenia. Tabulation of 13,240 improved cases. The three grades of improvement in percentage of total number of cases improved.

Chapuis and Georgi¹⁷⁷ report that better than 50 per cent of their total remission figures remained stable for from three to seven years, and—in line with the observations of Rupp and Fletcher²⁴⁸—that the full remissions held better than the social remissions: relapse rate for the latter was 42.8 per cent compared with 17 per cent for the former. They also found

that the duration of illness before treatment was begun bore no relation to the relapse rate.

It is of course important in this connection to consider the fact that the total overall period of hospitalization remains significantly shortened for the insulin-treated patients, even in those cases who do suffer relapses. Insulin coma treatment can return patients to the community in a markedly improved condition for a considerable period of time, even in those cases in which the remission does not remain stable. Shurley and Bond²⁵⁹ found that even the relapsed patients among the insulin-treated group spent an average of two months more at home than in the hospital, while the relapses among the control group spent seven and a half more months in the hospital than at home.

It should also be taken into account that the above relapse rates, with the notable exception of the studies of Horwitz et al.²⁰⁰ and Rupp and Fletcher,²⁴⁸ give no indication of the number of patients who again attain remission with a successive course of treatment. In this connection I have frequently observed that relapses after adequate deep insulin coma treatments (60 comas) are milder than the original illness, reflected not only in the clinical symptoms, but also in a less severe distortion of the response pattern of the autonomic nervous system. These relapses sometimes take the form of merely depressive or anxious episodes, which then respond to electric treatment, convulsive or nonconvulsive, while prior to the original insulin coma treatment, such patients had been unresponsive to electric forms of treatment or may even have been made worse by them (see pp. 43, 241, 243, and 244).

A certain number of apparent relapses turn out not to be actual relapses at all, but merely understandable reactions to problems of readjustment which are problems of the total family setting rather than readjustment problems of the patient alone. These must be met with understanding guidance including a wholesome psychotherapeutic influence upon the entire family group. This point emphasizes the importance of follow-up psychotherapy after otherwise successful physical treatment.

Now to the management of the genuine full-blown relapse of a schizophrenic illness. If this occurs within three months after a course of sixty deep insulin comas, or for that matter, if sixty deep comas fail to bring about a good clinical result, frontal lobotomy is indicated. The family should be prepared for such an eventuality, so that no unnecessary time is lost. The most important issue in the treatment of schizophrenia is prompt termination of psychotic episodes by any effective means available, since the amount of time over which the illness is allowed to ingrain itself upon the personality has an effect upon the ultimate chances for results of first-rate quality. Whatever the shortcomings of our methods of treatment, there are certainly enough tools in our hands to keep unsatisfactory results or relapses at a minimum. Of the schizophrenic patients whom I have treated with deep insulin coma during the past five years, not a single one remains hospitalized, although included in my series are cases who had been severely ill for as long as five years and who had become back wards patients of state hospitals. Those who relapsed late, after one to two years of social or complete remission, were promptly retreated—only one with another series of insulin comas; the others with whatever electric treatment method was indicated in consideration of their

now different autonomic reaction patterns. Those who relapsed early—within three months or less—were promptly subjected to lobotomy. Those who failed to achieve or to hold a good social remission after lobotomy either were reoperated on, or, if the first lobotomy had been fairly radical, were given from seven to thirty eight electric coma treatments of 7 to 10 minutes each. One of the two patients in the latter group—a former back ward state hospital patient—achieved a degree of recovery closely bordering on complete recovery. He is usefully employed and in every way his former own best self, except for his level of aspirations which has become more modest. The other patient, also a former back ward state hospital patient, has achieved a fairly satisfactory state of social remission; she lives at home and is quite congenial with her family. Her only disability is that she is somewhat demanding in her occupational and social relationships and hence changes jobs very often, the remarkable thing being, however, that she manages to obtain a new position not too long after each old one wears out.

In actual practice, therefore, there need remain few if any “hopeless schizophrenics,” provided one is prepared to use the full gamut and range of physical and psychologic treatment methods available.

Psychophysiologic Effects of Insulin Subcoma Treatment

Insulin subcoma treatment was initiated by Sargant and his co-workers^{287, 288} for the treatment of war neuroses associated with marked physical exhaustion and weight loss caused by the combined effects of physical fatigue and combat stress. Insulin subcoma treatment has proved successful in this particular constellation of physical and emotional exhaustion. After the war it was used in the treatment of neuroses in civilian life, and it has proved quite effective in properly selected cases, especially in certain reactive depressions associated with marked weight loss (D. E. Sands²⁸⁵) and in anxiety tension states (I. J. Sands²⁸⁶). In 400 cases of psychoneuroses, including basic anxiety states and cases of combat fatigue, total clinical improvement was achieved in 67.2 per cent, and complete and/or social remissions in 32.0 per cent of the total group (Tomlinson and Ozarin,²⁸⁹ D. E. Sands,²⁸⁵ Hohman and Kline,²⁸¹ Carnet et al.²⁷⁸).

I have used insulin subcoma for the treatment of reactive depressions in which there is a significant admixture of anxiety, as well as for the treatment of chronic anxiety states with pronounced epinephrine- or nor-epinephrine-triggered anxiety, particularly those with somatization reactions and psychosomatic symptoms, e.g., headaches, pylorospasm, spastic colon, etc. In patients who respond well to the treatment, the following psychophysiologic phenomena are usually observed: Half an hour after injection the patient will become upset and jittery. The anxiety thus released usually diminishes or disappears within one and one-quarter hour after injection, at which time depression generally comes to the fore. One of my patients described the depressive mood swing which characterizes the second half of the treatment as “a feeling of being sorry for oneself, all problems becoming magnified.” This phase lasts until termination of the treatment which is usually scheduled three hours after the injection of insulin. At this time the patient is given a glass of orange juice and a good appetizing meal. This is followed by a pleasant feeling of relaxation. As the same patient put it, “Then after you eat you are so interested in

stuffing yourself that the jitters and the depression leave you and then you relax—all the problems are still there but you just don't think of them and you feel fine. At the present moment (one and a half hours after interruption) everything is perfect since an hour ago." This is the time to give psychotherapy, which is usually accepted without significant defensive resistance. Patients who hitherto had offered frantic resistance to any psychotherapeutic effort, in this relaxed postsubcoma phase often came to look at their problems objectively and accept dynamic insight to which they had been entirely inaccessible before.

This has happened in a number of patients who had up to three years of intensive psychoanalytic therapy before being subjected to insulin subcoma. A recent example is a 31 year old woman who was suffering

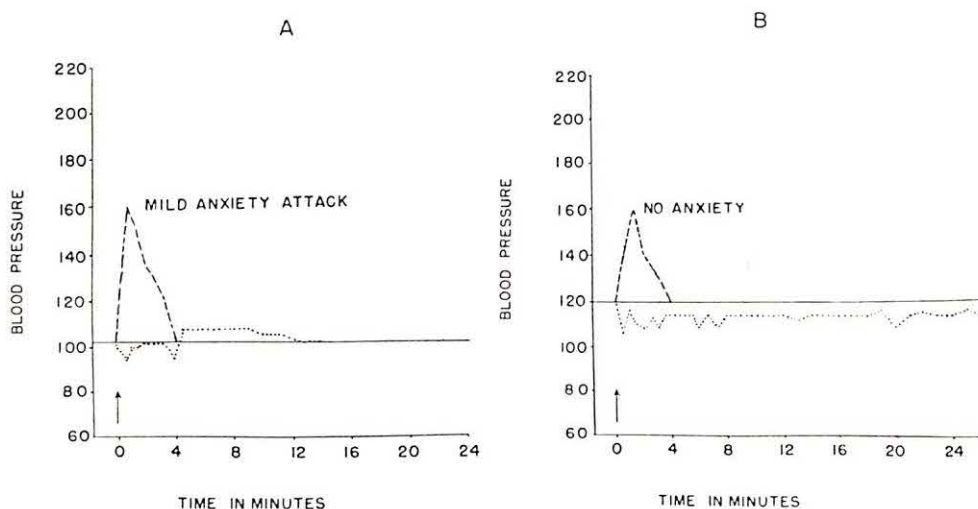


Fig. 62. Female, aged 47 years, suffering from anxiety tension state with depressive features, chronic and severe.

The broken line represents the blood pressure response to epinephrine (0.05 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Funkenstein test prior to treatment. Note slight epinephrine-precipitable anxiety, and slight and transitory fall of blood pressure after injection of mecholyl, followed by overshooting above the preinjection level.

B. Funkenstein test after 21 insulin subcoma treatments. Clinically the patient was free from anxiety, but mild depression lingered on at the time of this test. Note rise of basic blood pressure; reduction in response to epinephrine with absence of epinephrine-precipitable anxiety, but prolonged fall of blood pressure after injection of mecholyl.

from an anxiety-tension state with hysterical and depressive features of five years' duration, in which a somatization reaction in the form of headaches played a predominant role. Her headaches were of sufficient severity to warrant repeated electroencephalographic examinations and careful neurosurgical study including pneumoencephalogram and arteriograms, after three years of psychoanalytic therapy had failed to bring about improvement. After a series of insulin subcoma treatments combined with psychotherapy, she recovered from her anxiety state as well as from her headaches, within two months after her neurosurgeon had referred her to me.

The Funkenstein graph shows that insulin in subcoma doses actually

reduces epinephrine-precipitable anxiety, while at the same time it enhances somewhat the response to mecholyl (Fig. 62). This enhanced responsiveness to parasympathetic stimulation, presumably due to increased sympathetic tone, is similar to that produced by nonconvulsive electric stimulation treatment. Insulin subcoma treatment, however, is slightly more effective in reducing epinephrine-precipitable anxiety than nonconvulsive electric treatment, particularly in cases in which the anxiety state has become chronic. In severe cases of schizophrenia, insulin subcoma treatment is of little value except as a symptomatic sedative (Rennie,²⁸⁴ Bennett²⁷⁷). While some authors (Tomlinson and Ozarin,²⁸⁹ Polatin and Spotnitz,²⁸³ Cohen^{279, 280}) obtained good clinical results, it can be surmised from the context that the illnesses in those cases had been particularly mild, and of brief duration. Holt, Landau, and Vernon²⁸² compared results of coma and subcoma in parallel groups of schizophrenics and found striking differences in response to subcoma as compared to deep insulin coma, obtaining only 32 per cent clinical improvement with subcoma as compared to 70 and 80 per cent improvement in the groups that received deep insulin coma treatment in conjunction with electric forms of treatment.

Psychophysiologic Effects of Ether Drip Infusion Treatment

During ether drip infusion treatment the patients usually feel relaxed. While the treatment has no actual narcotic effect, some patients tend to fall asleep if undisturbed, especially toward the end of the treatment. A few may show slight euphoria or drowsiness. At the end of the treatment, however, all patients are fully awake, able to get up freely and move about without incoordination, although they usually feel more relaxed. The without incoordination, although they usually feel more relaxed. The claim has been made that the treatment gave specific relief from depressions, both the manic-depressive and involuntional types, similar to the relief afforded by electroshock. Ferraro and his coworkers²⁹⁰ reported in a small series of cases of affective disorders—16 manic-depressives and 38 cases of involuntional psychoses—significant therapeutic benefit in 87.5 per cent of the manic-depressive cases and 36.8 per cent of the cases of involuntional psychosis with an additional 42.08 per cent of involuntional psychoses showing some improvement. In Funkenstein's recent series²⁹¹ the significantly improved category seems to have leveled off at 52 per cent. While this improvement rate is significantly higher than that occurring spontaneously at a comparable phase of the illness (see p. 212), it is definitely less than that obtainable by electric treatment techniques and not higher than that obtained by psychotherapy alone (Tables 5, 9, 13) or reinforced by physical placebo treatments such as intravenous saline infusions (Funkenstein and Meade²⁹¹).

In 3 cases I was able to compare the efficacy of ether drip infusion treatment with electric forms of therapy. The first of these is that of a woman aged 57 who had recovered spontaneously within a year of onset from a depression which she suffered thirty years previously. The present depressive illness, complicated by significant admixture of anxiety, for which she consulted me was of seven years' duration without spontaneous let-up; it had become increasingly severe during the past year. Twelve ether drip infusion treatments administered between October 12, 1950 and October 26, 1950 brought about a good social remission. A year later she relapsed, and this time achieved a slightly better state of social remission—bordering

on complete recovery—after seven combined convulsive-nonconvulsive electroshock treatments.

The second case is that of an agitated depression of four years' duration in a male aged 53 years. Seventeen ether drip infusion treatments brought about only slight improvement, but sufficient to encourage the patient to try other forms of physical treatment. He finally achieved a satisfactory state of social recovery, bordering on complete recovery, after a total of twenty nine electric treatments, of which the first seven were nonconvulsive, the remaining twenty-two combined convulsive-nonconvulsive treatments.

The third case is that of a severe involutional depression of one and one half years' duration in a 50 year old man. He failed to respond to twenty-two ether drip infusion treatments, and relapsed within two weeks after achieving a temporary state of moderate but significant clinical improvement, still quite short of social recovery, after eleven convulsive electroshock treatments. He did not respond at all to a total of two years of psychotherapy. He is now, three years after onset, being considered for frontal lobotomy.

The clinical advantages of the technique are that the treatment is applied in circumstances of pleasant cooperation and the patient develops no anxiety in connection with the treatment; that during the treatment the patient experiences a sense of well being and throughout the treatment is in touch with the physician. A further advantage is that no confusion or memory disturbance associated with the treatment accrues, and that the thread of awareness is at no time interrupted. This is a particular advantage in patients with obsessive personality who fear and resent anything that interferes with their continued conscious control.

The physical advantages are that treatment can be given in older patients with hypertension, arteriosclerosis, bone deformities, or hernias, i.e., with practically no contraindications.

There are obvious psychotherapeutic situational aspects associated with the treatment. The diaphoresis, the diuresis, the sense of warmth, the feeling of relaxation—all part of a pleasant, mild, parasympathetic stimulus with the implication of recuperation and restoration of energies—create a psychologic atmosphere favorable to therapeutic effect. In the course of the treatment, patients become more talkative, more cooperative, feel more at ease with the therapist, and are more eager to discuss their personal problems, although there is no actual abreactive ventilation comparable to that elicited by ether inhalation or by the barbiturates. Narcosis does not play any part in the procedure, since the concentration of ether is far below the level required for narcosis. The total experience of this form of treatment is regarded as pleasant by the patients; and in those patients in whom the treatment fails to bring about a sufficiently good or lasting remission, or in whom it does not produce any significant improvement, it still has the virtue of conditioning the patients favorably to the acceptance of other more effective forms of physical treatment.

Psychophysiologic Effects of Frontal Lobotomy, Topectomy, Subcortical Undercutting, and Thalamotomy

Frontal lobotomy is capable of relieving anxiety, tension, hostility, obsessions, compulsions, paranoid projections, and depression; of abolishing

introversion, egocentricity, and ruminations with futile inquiries into the future; and of relieving those aspects of physical, particularly visceral, pain which constitute a threat, arouse fear, and cause persistent obsessive emotional suffering. Frontal lobotomy accomplishes these results at a price, since normal brain connections have to be sacrificed. This price is sometimes infinitesimal, indeed practically unnoticed and unnoticeable, being limited to a mere reduction in originality and creativeness. Many patients in whose lives originality and creativity never played an important role thus appear to have gained a great deal and lost very little. At the other end of the scale are the patients in whom lobotomy results in lasting severe apathy and inertia. In between are patients who exhibit, sometimes temporarily, sometimes permanently, varying degrees of childishness, tactlessness, and undisciplined social behavior. Since the operation tends to reduce anticipation and foresight it may reduce as a result the patient's tact. The operation deprives ideational acts of their ability to distress the patient, and this bleaching of the affect of ego cathexis reduces to a marked degree the patient's tendency toward introversion. Interest in his own feelings and sensations diminishes; interest in things outside himself increases. The operation reduces the aggressive drives that power personal ambition, although not in all cases. Intelligence, including biologic intelligence (Halstead²⁹⁸), is left intact. In general, realistic egotism replaces the former tormenting egocentricity of the patient. The need for creative expression subsides sometimes after a brief last flowering following the operation. The outward forms of social conformity are retained. Maternal instinct and maternal pride remain high. There is a certain lack of curiosity which is not easily measurable by tests.

Sexual performance, if disrupted by anxieties, compulsive fears, or obsessions prior to operation, tends to become restored to adequate technical proficiency after operation, but tends to remain rather sedate and uninspired, having lost its quality of urgency and its passionate aspects. In the case of a woman, in whom sexual responsiveness and performance had been adequate in spite of a severe and otherwise totally disabling compulsive neurosis which had remained resistant for years to analytic and other psychotherapy and periodic hospitalizations in mental hospitals, the sex act had to be regularly preceded by elaborate preparations of inspection and cleaning with which her presumably healthy husband obligingly cooperated. After her successful lobotomy by which she was completely relieved of her obsessions and compulsions without other overt personality deficit, both the patient and her husband lost a good deal of interest in sexual activity, although when relations were carried out on rather rare occasions, they were satisfying to both.

There is a report of one case in the literature³⁰⁰ in which increased virility with actual enlargement of the gonads has occurred after lobotomy.

A good many of the undesirable side effects of frontal lobotomy, especially apparent childishness, the lack of tact, and the undisciplined social behavior, can be overcome by subsequent guidance and psychotherapy. Freeman and Watts²⁹⁴ particularly stress the important role of the family during the period of convalescence. The family can be of great assistance in a way which almost amounts to helping the patient grow up again. This convalescence, or gradually growing up again, may take two or more years. Good paternal and maternal figures who will stand by the patient's

side are very important in this phase. This growing up again may start at a very primitive level and old forgotten behavior patterns from the dim past of the patient's own early childhood may temporarily come to the fore again. In one of my patients who was operated on at my suggestion for a schizophrenic illness unresponsive to any other form of treatment—a disease that was severe for five years, but had probably been coming on insidiously for more than ten years—the operation resulted in full social, occupational, and personal recovery after a stormy course of two and a half years in which the growing up process could be followed and aided in a progressively effective manner. During the postoperative period this patient went through a peculiar act of play. While supposed to be eating, he would stuff his mouth full of food like a squirrel, then spit it all out explosively as far as he could, laughing uproariously at the result. It so happened that on a Sunday when I was making rounds, his mother came in just at the height of the performance. She almost cried with joy, for there was her little boy again. This was exactly what he had done as a small child when he had first been taught to eat. This man is now holding a responsible job, is a considerate husband, and has been a tower of strength to his whole family in a recent emergency.

The high grade of occupational and personal rehabilitation attainable by frontal lobotomy is sometimes striking indeed. All of us have seen cases like Freeman and Watts'²⁹⁴ case 76 (pp. 220ff.), a successfully rehabilitated member of a learned profession. Remarkable qualities of leadership in social and community activities were preserved in another one of Freeman and Watts' cases, namely 42 (pp. 227ff.). This case is particularly remarkable, because loss of social sense (Kleist's³⁰¹ "social ego"), the holos function of the personality, as opposed to the ego function, is frequently impaired after lobotomy. Freeman and Watts'²⁹⁴ contribute a case which attests to the preservation of high morale during wartime (case 69). They also report the interesting case of a successfully rehabilitated school teacher. Her high skill in dealing and empathizing with children was enhanced rather than detracted from following operation. Freeman and Watts report that no physicians, dentists, or nurses have been successfully rehabilitated. These are professions in which the qualification of foresight is a particularly crucial one.

In the early days, of course, the outcome of lobotomy was rather unpredictable. There was an element of gambling about it, as if one were about to make a drawing in a lottery, which made it a difficult decision. In the meantime, important observations concerning the influence of the extent of the operation—perhaps not quite yet classifiable as a dawning knowledge of the specific functions of specific tracts—as well as the plan to carry out the operation in several stages have gradually introduced an element of somewhat greater control of results.

An observation of outstanding importance was made by Freeman and Watts in the course of their work on cases of pain without mental disease. They found that frontal lobotomy takes away more from a normal individual who has pain only than from an individual who is mentally sick. For this reason they advocated and carried out less extensive operations in such cases. They found that sectioning of the orbital sector alone was sufficient for the relief of pain and thus they were able to prevent undesirable side effects in these cases. This observation indicated an inverse

quantitative relationship between the appearance of undesirable side effects after the operation and the amount of emotional and mental abnormality before operation. This is of profound significance in supplying a lead for promising attempts to avoid side effects by tailor-making the extent of the operation proportionally to the severity and resistiveness to treatment of the mental illness to be relieved. When lobotomy is carried out in a relatively mild illness, excessive depletion, apathy, and inertia are more likely to occur than when the same operation is carried out in a very severe or resistant illness. This emphasizes the importance of adequate therapeutic testing of the severity of the disease as a prerequisite for proper evaluation of its severity prior to the lobotomy. The practical safeguards to be taken in this direction will be dealt with in greater detail in the chapter on indications (XIII).

The problem of the downgrading of the patient's personality is, of course, most portentous. In a number of fortunate cases this downgrading remained so slight that it turned out to be merely a theoretical and academic postulate, difficult to prove in spite of careful observations and tests; for others, less fortunate, it became a sad actuality. Partridge,³⁰⁵ who has contributed a most scholarly and penetrating book on the subject, formulates this problem succinctly: "It is this reduction of the psychic life to a simpler level that makes us hesitate before recommending operation. Post-operatively the responsiveness of the patient is reduced, and in so far as spontaneity, so-called, is a measure of responsiveness to stimuli, so is that also reduced. There is less activity and more inertia. There is a blunting of affect, due to a reduced complexity and intensity of feeling, and therefore there is less variation and more equability of temperament. The intellectual processes are simpler, with attention to the immediate rather than to the remote, to the factual rather than to the theoretical, with decisions that are simple rather than deliberative, and with a restriction of the intellectual range. The total pattern of reaction is simpler, marked by an essential tendency towards avoiding discomfort and courting pleasure, with lowered standards of criticism, reduced self-awareness, and diminished self-control, which last is considerably offset by the lessening of responsiveness in general."

And yet, Partridge suggests, "there are other things to be considered. When one sees a patient who has been insidiously ill for 8 years, and severely so for between 3 and 4 years, with an increasing deterioration which culminates in the eating of feces while in the padded cell, and sees him just over a year after operation, physically robust, and singing excellently in a well-known choir which is participating in a choral competition, having taken the bus from his home to get there; when one sees a patient with contractures of the semi-tendinosus and semi-membranosus from having spent the better part of 7 years in fixed attitudes of prayer between bouts of being violent, with double incontinence, and sees him within a year of operation making 50 at cricket (equivalent to two home runs in baseball), having taken the afternoon off work to play for a local side, one feels bound to make some inquiry as to how it is done." This question, simply put, "how it is done," is indeed the crucial question.

Tests in general have been disappointing, and have not supplied an answer to the crucial question posed by Partridge as to "how it is done." There was evidence that a decline in score with the Porteus³⁰⁹ maze test

after frontal lobotomy was correlated with clinical improvement. This correlation, however, did not hold for cases of minimal, for instance, transorbital lobotomy, in which there was no decline in the Porteus maze test, in spite of clinical success of the operation. Freeman and Watts²⁹⁴ came to the conclusion that the standard intelligence tests, as well as the Porteus maze test, merely may reflect incidental effects of brain damage, and have no connection with the basic therapeutic effect of the operation. They believe that the outstanding effect is a primary change of attitude towards the self. In order to explain this change adequately, Freeman and Watts found it necessary to revert to the old concepts of faculty psychology. The faculty which they considered lost was that of "self-continuity," expressed by the "self-regarding span." In order to test this fact they designed, in collaboration with Robinson,^{294,310} the self-regarding span test augmented by a sensibility questionnaire.

However, I feel that their results could be much better defined in dynamic terms of defensive operations. I found in my cases that the defensive operations of isolation and denial appear to play a particularly striking role following lobotomy, and I found these observations confirmed by the descriptions of and the conclusions of other observers. Freeman and Watts have supplied excellent case histories proving that not merely a palliation of the disturbance was achieved but that recovery of the patient was associated with real insight and also strengthening of defensive operations, particularly isolation and denial. Freeman and Watts' case 465²⁹⁴ (p. 142) shows most dramatically the emergence of the defensive force of isolation. Freeman and Watts state: "Then as the last fibers are severed on the second side he will abruptly lose contact and deny that an operation is being performed even while he can still feel the needle pricks and sutures. . . . After the operation was over, the patient denied that he had had a lobotomy. 'Oh, yes,' he said at the time of his discharge, 'you put me on the operating table, and shaved my head, and cut down on the skull, and drilled some holes on each side, and I remember feeling a pain behind my eye and seeing my little niece, and trying to talk to her; but that's where you stopped. You didn't go on operating, you just closed up and put the bandage on. You couldn't have operated on me because I'm just the same as I was before, and there would be some change if you had operated on me.' The authors add that "these statements, from a very intelligent and articulate individual, give an indication of profound disorientation with regard to self." I feel that this disorientation in regard to self in an obviously conscious person is much more akin to the mechanisms of isolation and denial than to actual disorientation. Freeman and Watts supply additional evidence of this in a conversation with the same patient sometime later.

"Doctor:	Has there been any change in you?
Patient:	None that I know of.
Doctor:	Were you upset when you came here?
Patient:	Yes, I was upset.
Doctor:	Are you upset now?
Patient:	No.
Doctor:	Is that a difference?
Patient:	Yes.
Doctor:	Did you want to kill yourself?

Patient: Yes.
Doctor: How about now?
Patient: No, I don't want to kill myself.
Doctor: Is that a change?
Patient: Yes.
Doctor: Did you think you could never get well?
Patient: Yes, I thought so.
Doctor: What about it?
Patient: I'm getting along all right.
Doctor: Now let's get it straight: you were upset, wanted to kill yourself, and thought you could never get well. Remember that? What is the change that has occurred in you?
Patient: None that I know of."

This certainly is an example of completely successful defense with the aid of the mechanisms of isolation and denial.

Another case which Freeman and Watts²⁹⁴ describe bears out this fact (case 478). This was an army nurse "who had been under treatment for a schizophrenic psychosis with extreme tension, first for two years in the Army, and then for more than a year in St. Elizabeths Hospital. Her tension was so extreme that she required six special nurses, two at a time, to control her homicidal and suicidal impulses. In addition, she had to be kept in a pack or a tub for several hours almost every day. When interviewed two months after operation, she was agreeable and smiling, remembered episodes when she had caught mice with her bare hands and eaten them alive, when she had broken her nurses' glasses, broken windows and so on, but when she was asked about the numerous scars that disfigured both wrists, she insistently replied: 'Oh, those don't mean anything. I must have scratched a mosquito bite sometime.' When she was reminded of the way in which she had cut deep gashes in her wrists, and then torn the wounds open in a frenzied effort to die, to infect herself, to make other people kill her, she admitted in rather awkward fashion that she had done these things, but it didn't matter now because the operation made her a good girl."

I feel that Freeman and Watts' concepts of disorientation of the self, disturbance of self-continuity and of self-regarding span, are manifestations of the defensive operation known as isolation, with or without denial. The same may apply also to the fact first observed by Freeman and Watts that hallucinations and other abnormal ideas—while still persisting—"lose power" after lobotomy. This could also be due to the fact that the defensive forces then succeed in isolating them. The process of isolation is similar to the process of concentration upon a major practical task at hand. Isolation is the removal from attention or concern of a painful or harrassing problem, a "stripping of its associative connections" definable as a "symbolic amnesia" (Freud⁶⁹). It is akin to the normal capacity for concentration and ranges from the ordinary disregarding of a toothache while doing an important piece of work to the isolation of a conflict within the confines of a ritualistic act performed by an obsessive neurotic. It may be significant that lobotomy, which particularly aids the defensive forces of isolation and denial, is especially effective in obsessive and compulsive neuroses, diseases in which the patients spontaneously attempt to relieve their suffering by the defensive maneuvers of isolation and denial. It

seems that these two defensive operations are particularly and specifically aided by frontal lobotomy.

Statistics of Therapeutic Results. The statistical data on overall clinical results are impressive. Fulton,²⁹⁵ who presided at the December meeting of the Association for Research in Nervous and Mental Disease in 1947, stated that the results of that symposium "can be briefly summarized by saying that they are highly impressive from a therapeutic standpoint, especially when considered in relation to the prognosis of the type of case on which the lobotomies have been carried out: 35-50 per cent of the hospitalized cases were reported as being sufficiently improved to be sent home to their families; 15-20 per cent of the remainder in the various series were sufficiently improved to be able to take on odd jobs within the institution; 25-35 per cent were unimproved, and not more than 1-3 per cent were said to have been made worse. The mortality rate is less than 1 per cent."

The results in Freeman and Watts'²⁹⁴ 600 cases indicate that about one-third of all patients are considered recovered; one-third improved; and one-third unimproved. Divided as to occupational status and residence, the results show that approximately 50 per cent of all patients are usefully occupied and live at home, another 25 per cent live at home but are not usefully employed. Only 25 per cent still require hospitalization. Of involutional psychotics and neurotics, 70 per cent are usefully occupied after lobotomy. Specific categories of improvement in specific disease groups are reported as follows: in schizophrenia—good results in 35 per cent, fair results in 38 per cent of the cases; in involutional psychoses—good results, 60 per cent, fair results, 24 per cent; in obsessive and other neuroses—good results, 57 per cent, fair results, 28 per cent. One of the most interesting findings is that in schizophrenics hospitalized for more than ten years prior to operation, the results are still good in 30 per cent of the cases, and fair in another 30 per cent.

The Connecticut Lobotomy Committee, composed of Moore, Friedman, Simon and Farmer,³⁰⁴ reported on a study of 200 cases with a follow-up of from three months to one year. Of these 200 cases, schizophrenia accounted for 163, manic-depressive psychoses for 10, involutional psychoses for 8, and psychoneuroses for 4. At the end of the year they found that 35 per cent of all cases were slightly improved, unimproved, or worse; 27 per cent moderately improved; and 38 per cent markedly improved or symptom-free. These figures represent a higher percentage in the last group than was found at the end of the three-month period following operation. In terms of environmental and work adjustment at the end of the year following operation, 48.8 per cent were at home. Of this group 59.2 per cent were working either full time or part time.

Greenblatt, Arnot, Solomon, and their coworkers²⁹⁷ reported results of lobotomy in the following categories: schizophrenia (*dementia praecox*), simple type, good results, 41 per cent, fair 24 per cent; catatonic type, 6 per cent good, 37 per cent fair; paranoid type, 6 per cent good, 16 per cent fair; hebephrenia, 6 per cent good, 6 per cent fair. In manic-depressive psychosis, depressed, results were good in 53 per cent, fair in 34 per cent; in chronic manic cases results were good in 20 per cent. In involutional psychoses results were good in 92 per cent. In "other paranoid psychoses" results were good in 25 per cent, fair in 50 per cent. In obses-

sive-compulsive psychoneurosis results were good in 43 per cent, fair in 57 per cent; in hypochondriac neuroses (3 cases) results were good in 100 per cent.

Variants of Operation and Results. Mettler and his coworkers³⁰³ produced similar results by cortical excisions of areas 9, 10, and/or 46 with less undesirable side effects in terms of inertia but with a postoperative clinical course more frequently complicated by epileptic seizures. Cortical undercutting such as introduced by Scoville and his coworkers³¹³ combined the favorable effects of cortical excisions with the absence of significant disturbance by epileptic seizures; the latter advantage is generally characteristic of operations limited to the white matter. These undercuttings so far appear to indicate that there is little specificity of the frontal lobes in their therapeutic effect on the psychotic disturbance but definite specificity in their effect on the undesirable side effects on personality. The psychoses were favorably affected by isolation of a quantitatively sufficient area of the frontal lobes irrespective of specific locality. There was little if any blunting of the personality following isolation of the orbital surface, but definite blunting occurred upon isolation of the superior surface of the frontal lobes. The latter was similar to but less in degree than that found following standard lobotomy. Scoville and his coworkers confirmed Freeman and Watts' finding that there is more apparent personality deficit in nonpsychotic than in psychotic patients following any type of lobotomy; hence, only fractional lobotomy should be used in patients suffering from mood disorders, neuroses, or pain. Among 118 cases of schizophrenia followed up for three months to two and one-half years, these authors found that after undercutting of the superior surface, which was carried out in 33 cases, results were good in 30 per cent. After undercutting of the orbital surface, carried out in 28 cases, results were good in 50 per cent. After undercutting of the medial surface including the cingulate gyrus, which was carried out in 32 cases, results were also good in 50 per cent, while in a control series of 25 cases of standard lobotomy, results were good in 40 per cent. The results in the affective psychoses, psychoneuroses, psychopathic personalities, and organic psychoses were still too small to allow significant statistical evaluation. Levine, Greenblatt, and Solomon³⁰² have recently established significant superiority of bimedial operations over the standard ones. These bimedial operations consisted in sectioning the medial white matter only, including its upper and lower parts, thus sectioning the cingulum fairly completely, as well as the medial part of the uncinate tract, but only the lower medial extension of the thalamofrontal tract.

Bimedial operation reduces psychotic symptoms as thoroughly as standard operation, but appears to give more satisfactory overall results by causing less "blunting," leaving the patient with more initiative, better socialization, and better preservation of emotional warmth and capacity for rapport.

Patients improved following bimedial operations also show the maximal degree of reduction of abnormal over-responsiveness to parasympathetic stimuli obtainable by any frontal lobe operation (as demonstrable by the Funkenstein test), thus indicating that bimedial section is the decisive element in bringing about restoration of normal autonomic reactivity after lobotomy.

Spiegel and his coworkers³¹⁴ obtained similarly favorable effects on psy-

chotic symptoms and reduction of undesirable side effects by thalamotomy. Of the pathologic symptoms, particularly nervous tension, anxiety, depression, irritability, agitation, hallucinations, and compulsions were relieved. Of 8 patients with 5 lesions in the dorsomedial nucleus of each side of the brain, each lesion measuring 3 mm. in diameter, 5 such lesions covering about one-seventh of the total volume of the dorsal medial nucleus of the thalamus, 4 were able to return home and 1 of them at the time of the report was about to find employment. Only 1 patient had impairment of memory and 4 some diminution of initiative. One of these had been subjected to frontal lobotomy two years prior to the thalamotomy so that this condition was probably due to the combined effect of these two procedures. This latter patient was also careless in his personal appearance. None of the other patients had developed any undesirable side effects such as those frequently occurring after frontal lobotomy, apart from transitory incontinence of the bladder, which occurred in 4 of 8 thalamotomized patients reported.

Psychophysiologic Effects of Surgery of the Temporal Lobe

Temporal lobectomy was first performed by Bailey and Gibbs²⁹² based on Gibbs'²⁹⁶ discovery of the specific role of the temporal lobe, especially its anterior portion, in the impulsive, violent acts of psychomotor epilepsy.

Psychomotor or temporal lobe epilepsy is characterized by trance-like attacks and confusional episodes. When such psychomotor seizures occur in individuals without convulsions, erroneous diagnosis of hysteria or schizophrenia is common. Fifty per cent of the patients with psychomotor or temporal lobe epilepsy have severe disorders of personality and one-half of these have a history of major psychosis. In contrast to this finding in temporal lobe epilepsy seizure activity in areas other than the temporal lobes is not commonly associated with disorder of personality or psychosis. Gibbs²⁹⁶ found that the nonictal psychiatric manifestations that occur in psychomotor epilepsy appear to be antithetic to the psychomotor attacks, that is, they seem to increase when the attacks diminish in frequency. These nonictal psychiatric symptoms can take many forms, covering almost the entire spectrum of psychiatric symptomatology; schizoid, paranoid, and manic-depressive pictures are common. Bailey and Gibbs²⁹² were encouraged by the therapeutic results obtained through the removal of the anterior portion of the temporal lobes, but they felt that their case material was not yet numerous enough nor observed over sufficiently long periods to allow final conclusions.

The possible relation of the temporal lobes to the symptoms of schizophrenia were suggested by the important stimulation studies of Penfield and Rasmussen,³⁰⁶ in which the activity of the temporal cortex was found to be important in influencing the quality and character of sensory perceptions, internal and external, as well as their interpretations, and to play an important part in eliciting memory reproductions, dreams, and hallucinations. Stimulation of the temporal cortex brought about illusions of distance, intensity, familiarity, strangeness or absurdity of sensory perceptions, auditory and visual, as well as phenomena of altered "ego scrutiny," alterations in the individual's opinion regarding his relationship to his environment or the relationship of his personal self to his own body, including a feeling as if his ego were removed from his body, which Pen-

field and Rasmussen call "illusions of introspection." These authors were also able to bring about dreams and hallucinations. These usually contained elements of reproductions of remembered experiences, were visual and/or auditory, and were of a progressively changing character, the action going forward as in a dream, in the course of which the patients sometimes became frightened. Penfield and Rasmussen found that the two temporal lobes are of equal value in the elicitation of these phenomena. One of them can be removed without evidence of marked memory loss or interference with the capacity for perceptual interpretation.

Subsequent operations on the temporal lobes (Thompson and Walker³¹⁵) have as yet proved inconclusive. But Williams and Freeman³¹⁶ report an important case of relief of a chronic hallucinosis following bilateral excision of the amygdaloid nucleus of the temporal lobe.

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INDICATIONS FOR PHYSICAL TREATMENT, BASED ON PSYCHIATRIC AND PSYCHOSOMATIC CLINICAL FINDINGS

Indications for specific treatment methods resolve themselves into two important steps: first, to decide whether psychologic treatment methods alone should be employed or whether physical treatment should be employed primarily; and secondly, if physical treatment has been decided on, to choose which type of physical treatment should be used.

Psychologic vs. Physical Methods. In evaluating a mental-emotional illness, the first aspect of the disturbance that engages the attention of the examiner is the nature of the affective disturbance—whether there is tension, depression, anxiety, fear, bewilderment, confusion, elation, and so on. This set of data alone of course can not determine the type of treatment to be given. The next step in the series of observations is directed toward the dynamics of the disturbance in which the behavior and attitude of the patient as well as the content of the disturbance play a role. The question to be decided is whether the patient is struggling—and against what or whom—or whether he is withdrawing or regressing, or whether he is punishing himself by turning the aggression inward. These observations alone likewise cannot determine the type of treatment to be used, but the degree to which *insight into the dynamics can be made meaningful to the patient and can enable the patient to participate in reality-testing under the therapist's guidance* is of prime importance in deciding whether psychologic treatment alone will be sufficient or whether physical treatment should be resorted to. The clarity of the therapist's understanding of these dynamics is prerequisite, but that alone of course does not help the patient to make use of the therapist's insight. A time limit must also be established. To carry out psychotherapy with psychotic patients over periods of years in the hope of eventually establishing fruitful interpersonal rapport, as in eight years spent with hospitalization and treatment in a case reported by Frieda Fromm-Reichmann in her book, while of great theoretical interest, has no or only greatly limited practical value. First of all, eight years represent a significant portion of a person's life, which when spent in illness is an irretrievable loss, and secondly, chances for an ultimately good grade of recovery become lessened the longer the illness pattern is allowed to ingrain itself upon the personality. I believe that if some response—in a totally disabling condition—is not obtainable by psychotherapy within a month, and in the case of a nondisabling condition within a year, further continuation of psychotherapy will not meas-

urably improve the chances for ultimate response, and a change of therapeutic approach is indicated.

The third item of importance in gathering data for our decision is the nature of the defensive operations. Effective psychotherapy requires a reasonable degree of *flexibility of defensive operations*. If the defensive operations are too intensely alerted, the therapeutic process will be difficult, but the experienced psychotherapist will be able to attain particularly gratifying responses in this group of patients. On the contrary, when defensive operations are at a low ebb, either broken down or exhausted, rapport can sometimes be established with remarkable ease, but the therapeutic progress will be slow or not forthcoming at all if psychologic methods alone are used. This is particularly true for depressions and other severely regressive psychotic states. Inadequacy of the defensive operations is of course also an important consideration in the institution of protective measures necessary for the prevention of suicide. Retained capacity for the healthy defense of identification, however, is always a good prognostic sign for results obtainable by psychotherapy, even if it occurs in conditions with otherwise poor prognostic connotations.

The fourth point influencing our decision is that of the general prognostic group to which the patient belongs. The element of diagnosis plays a role in this respect only. It is important, however, to accept the fact that the general diagnostic criteria are superseded by conclusions derived from the unique constellation of the dynamics of the illness and by the patient's capacity for improving his defensive position—by his residual capacity for normal defenses, by his capacity to improve his defensive position under psychotherapeutic guidance and with the aid of effective reality testing, and by his general capacity for meaningful communication with the therapist. If the patient scores low in all these respects, physical treatment should be considered.

If the patient has good capacity for meaningful communication with the therapist, if the therapist's insight into the dynamics can be made meaningful to the patient, if the patient can participate in reality-testing under the therapist's guidance, and if the defenses are either reasonably flexible or not too intensely overalerted, fruitful psychotherapy is possible. If, on the other hand, the patient's general capacity for meaningful communication is low, if insight into the dynamics cannot be made meaningful to the patient, if the patient cannot participate in reality-testing, and if the defenses are either down, exhausted and inactive, or so intensely alerted and overactive that they cannot be overcome, psychotherapy is unlikely to succeed within a reasonable time limit and physical treatment is indicated.

Choice of Physical Method. Once the decision to use physical treatment has been made, the type of physical treatment to be employed must be determined. This choice again depends upon four factors, namely: (1) the nature of the basic mood component of the condition to be relieved; (2) the state of the defensive operations; (3) the autonomic response pattern, indicating whether there is abnormality of reaction to sympathetic or parasympathetic stimuli or both (Funkenstein¹ test); (4) last, and least important, the diagnostic category. The first three of these sets of factors I have conveniently summarized in Fig. 63 which constitutes an attempt at a schematic psychophysiologic graph.

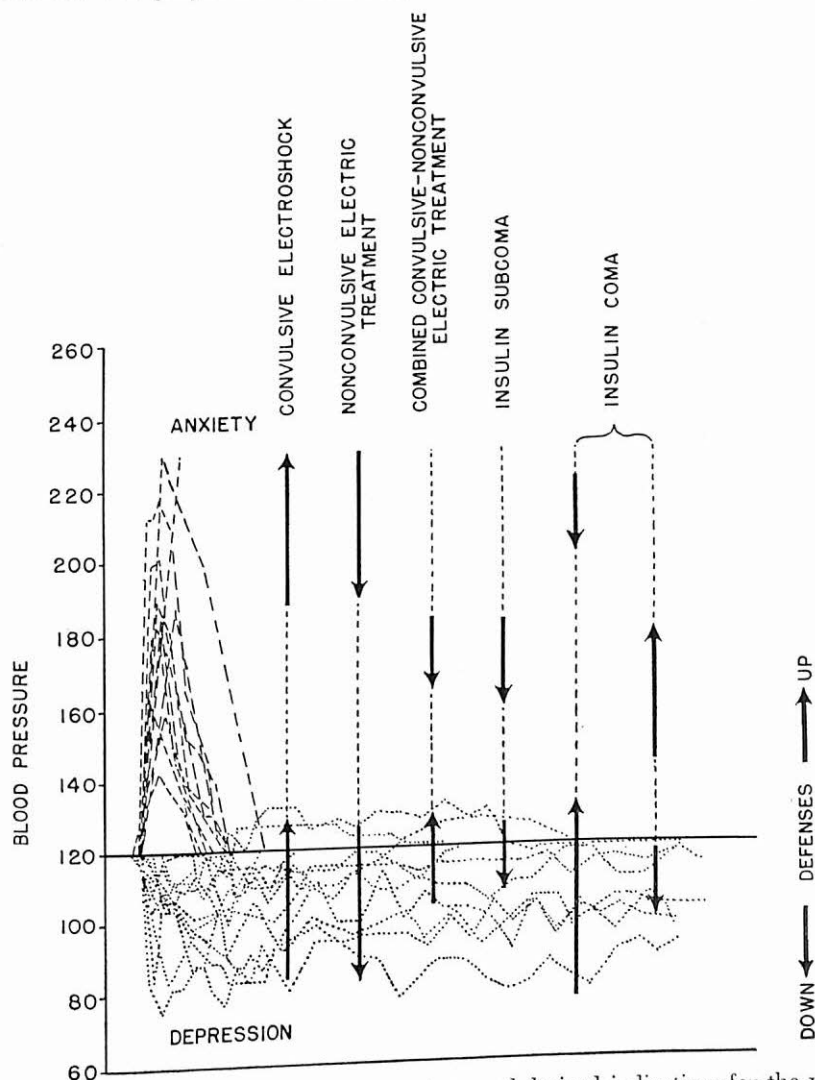


Fig. 63. The psychophysiologic response pattern and derived indications for the various physical treatment techniques. The basic components of this graph are the varying types and degrees of blood pressure responses to epinephrine (broken lines with long dashes) and to mecholyl (dotted lines). Epinephrine injection causes varying degrees of elevation of the blood pressure, and may precipitate anxiety. Mecholyl injection causes varying degrees of lowering of the blood pressure; marked and prolonged lowering of the blood pressure by mecholyl is characteristic of depression. The defenses go up with anxiety and down with depression.

A general shift upward on the graph indicates lessening of depression, lessening of blood pressure fall on mecholyl injection, increased reaction to epinephrine, alerting of the defenses, and finally, possible precipitation of anxiety. A shift downward indicates relief of anxiety, lessening of reaction to epinephrine, increase and prolongation of blood pressure fall on mecholyl injection, lowering of defenses, and finally possible precipitation of depression.

The way in which the various types of physical treatment alter these psychophysiologic constellations is indicated by the solid arrows (connected by short-dashed interrupted lines).

Convulsive electroshock treatment causes an upward shift on the graph. i.e., the treatment relieves depression, decreases and shortens the blood

pressure drop on mecholyl injection, increases response to epinephrine, reerects defenses, but may precipitate anxiety. Convulsive electric treatment is indicated whenever there is depression (or other dynamically equivalent or related regressive manifestations), marked and prolonged blood pressure fall on injection of mecholyl, and exhaustion or inactivity of defensive operations; but convulsive electroshock treatment is contraindicated when there is epinephrine-precipitable anxiety.

When there is depression as well as exhaustion or inactivity of defensive operations, convulsive electroshock therapy may even be successful when the response to mecholyl is not grossly abnormal. In other types of regressive psychotic states, however, convulsive electroshock is greatly reduced in effectiveness whenever blood pressure fall on mecholyl injection is neither enhanced nor prolonged.

Nonconvulsive electric treatment causes a downward shift on the psychophysiologic graph, i.e., it relieves anxiety, reduces response to epinephrine, enhances and prolongs the fall of blood pressure after injection of mecholyl, lowers the defenses, and may precipitate depression. Nonconvulsive treatment, therefore, is indicated when there is epinephrine-precipitable anxiety and when defensive operations are overalerted, but contraindicated when there is depression, when defenses have broken down, and when there is an excessive and prolonged blood pressure fall on mecholyl injection. The same is true for insulin subcoma treatment, except for the fact that the latter causes a less marked enhancement and prolongation of the mecholyl response and correspondingly is less likely to lead to precipitation and aggravation of depression.

Combined convulsive-nonconvulsive electric treatment, as well as insulin coma treatment, does not produce a unilateral shift on the psychophysiologic graph, but changes the response pattern in the direction of homeostasis. Combined convulsive-nonconvulsive electric treatment, as well as insulin coma treatment, is capable of relieving regressive psychotic manifestations, including depression, of reducing and shortening the blood pressure fall on mecholyl injection but, at the same time, is also capable of relieving anxiety and reducing excessive response to epinephrine, i.e., epinephrine-precipitable anxiety. With these types of treatment, the defenses also undergo a more balanced process of reorganization which, with combined convulsive-nonconvulsive electric treatment, consists first in a reforming of defenses followed by a reorganization in terms of greater flexibility; and in the case of insulin coma treatment, first in a lowering of defenses, followed by a constructive reorganization. Combined convulsive-nonconvulsive electric treatment or insulin coma treatment, therefore, is indicated whenever there is a combination of depression or other regressive psychotic manifestations associated with enhanced and prolonged blood pressure fall on mecholyl injection on the one hand, with epinephrine-precipitable anxiety on the other.

Only insulin coma treatment is capable of enhancing both the responsiveness to adrenergic stimulation, as manifested by the blood pressure rise on epinephrine injection, and simultaneously the responsiveness to cholinergic stimulation, as expressed by blood pressure fall on mecholyl injection, whenever both these responses were underactive before treatment. This cannot be done with any known form of electric treatment. Inadequate response to epinephrine as well as to mecholyl is found only

in schizophrenic psychoses; it constitutes, for the reason given above, an absolute indication for insulin coma treatment.

Insulin coma treatment may also be effective in schizophrenic states if there is no gross abnormality of the autonomic response pattern, although the statistical likelihood of good clinical response is lessened in such cases as compared to those in which the autonomic response pattern is grossly abnormal.

It is my considered opinion that lobotomy in cases of mental illness is only indicated when adequate amounts of other forms of physical treatment have failed. This view has also been emphasized by Kalinowsky and Scarff.³ The only exceptions in my opinion are patients suffering from severe intractable obsessive-compulsive states who have failed on adequate psychotherapy and who for physical or other reasons are regarded as unsuitable for a prolonged course of combined convulsive-nonconvulsive electric treatment or of insulin coma treatment. It is important to keep in mind, however, that such seemingly intractable obsessive-compulsive neuroses sometimes respond—in the younger age groups—to sixty deep insulin comas and—in the middle and older age groups—to courses of sixteen to twenty combined convulsive-nonconvulsive electric treatments. In some patients with obsessive-compulsive states, regardless of age, a series of nonconvulsive electric treatments alone may be successful provided the patients belong to the rare group in whom there is not only excessive response to adrenergic stimuli associated with epinephrine-precipitable anxiety, but in whom injection of mecholyl also produces a rise in blood pressure instead of a blood pressure fall—i.e., in those patients in whom a maximal upward shift on the psychophysiologic graph has taken place before treatment.

There are three reasons why it is important to insist on adequate pre-treatment by other methods before lobotomy. First of all, since some risk of psychic loss is practically inevitable, a definitive surgical procedure—implying such risk of lasting undesirable side effects—should not be carried out as long as there is the possibility, if not the probability, that other procedures implying less risk may bring about an otherwise equally satisfactory therapeutic result.

Secondly, it has been found that the likelihood of occurrence of undesirable side effects following the operation—such as excessive depletion, apathy, or inertia—is inversely proportional to the degree of severity of the mental disturbance to be relieved by the operation. In other words, the result of a lobotomy is likely to be better in cases which have resisted all other forms of treatment than in those which might have been relieved with other forms of treatment as well.

Thirdly, the intensity of the illness as tested by treatment resistance also permits the team of neurosurgeon and neuropsychiatrist adequately to gauge the extent of the operation to be performed.

Using the degree of resistance to nonsurgical methods as a guide in planning the extent of the lobotomy to be performed has been of great value. An outstanding example has been the case of a severe involutional depression which had been utterly resistant to three courses of convulsive electroshock treatments totalling fifty-three treatments which produced only negligible transitory effects. This fact made it reasonable to recommend a maximal standard lobotomy. The therapeutic effect of this operation

was excellent, resulting in practically complete recovery without recognizable undesirable side effects. I am sure that such an extensive lobotomy might have been excessive in cases of less treatment-resistant illnesses. In this case, however, I am sure that any less extensive lobotomy would have yielded unsatisfactory and inadequate results. The minimum standard for testing treatment resistance prior to lobotomy in cases of involutional psychosis or of chronic manic-depressive psychosis is—in my opinion—a course of twenty electroshock treatments; in cases of schizophrenia, sixty deep insulin coma treatments. If these courses of treatment yield inadequate results or if relapse occurs within less than two to three months, lobotomy is indicated.

In those cases of involutional psychosis or of chronic recurrent manic-depressive psychosis in old people in which relapse occurs later, but in whom another course of electroshock treatment would constitute undue physical risk, I prefer lobotomy to a second course of electroshock treatments. The same applies also to those cases in whom maintenance electroshock treatment (Geoghegan,² Stevenson and Geoghegan⁴) is not feasible for the same reason.

I also prefer lobotomy to a second course of convulsive electroshock treatments in those relapsed patients in the older age groups who reacted to convulsive electroshock as well as to combined electric treatments with build-up of anxiety, and whose obsessive personality trends make them particularly resentful of marked and troublesome memory disturbances brought about by the maximal amounts of alternating current necessary in these cases in order to achieve any therapeutic effect.

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XIV

PRACTICAL TREATMENT TECHNIQUES

PSYCHOTHERAPY

Hypnosis

The reader may be surprised to find the discussion of the techniques of psychotherapy beginning with hypnosis, which is perhaps the least used method of psychotherapy today. The reasons for doing so are historical as well as didactic. Modern psychotherapy is directly derived from the techniques, observations, insights, and theories which developed out of the experiences with hypnotic therapy in the nineteenth century. From the didactic point of view, the doctor-patient relationship in hypnosis is of such clear-cut simplicity and the setting so unequivocal that it may serve as a useful point of departure for a discussion of techniques of psychotherapy. Starting with the technique of hypnosis we shall then work up to the more complicated procedures.

Modern psychotherapy is based on the recognition of the distinctness and separateness of the major institutions of the mental apparatus: the desires, with their biologic drives and urges (the id) on the one hand, and the ego with its conscience (the superego) on the other. The distinctness and separateness of these institutions was first observed by Breuer and Freud¹⁵ in patients under hypnosis, and hypnosis still remains the most clear-cut means of demonstrating their distinctness and separateness today. The reason for this fact is that in hypnosis the physician takes over some of the important functions of the patient's ego, thus exposing the independent existence of the other institutions of the mental apparatus, mainly the id and to some extent the superego. The superego is more intimately connected with the ego and is probably merely an unconscious extension of the ego itself. Furthermore the superego is an extremely labile part of the mental apparatus, being continuously dependent upon, in fact parasitic on, prevailing external social-moral forces, values, and attitudes. Foremost among these are religion, law, and public opinion. This was brought home to me in a vivid manner during my study of war crimes.²

Be this as it may, the superego has to be reckoned with in hypnosis as that part of the ego which is more dependent on general social constellations than on the doctor-patient relationship per se. Furthermore, it is important to realize—standard psychoanalytic opinion to the contrary notwithstanding—that in hypnosis the patient's ego functions are not entirely suspended and taken over by the physician, not even in deep hypnosis. An inner, reserved core of the ego, as it were, retains its capacity

and watchfulness in the form of a mental reservation which may at any time spring to the rescue or run interference, whichever way one looks at it, and either wake the patient up or throw him into a deeper trance in which contact with the hypnotist is lost.

Even in deep hypnosis, therefore, the ego yields only as much control to the therapist as it chooses to. It almost always allows itself to be assuaged to revoke the inhibitions of those parts of the motor apparatus which were inhibited because of their actual or potential participation in the forbidden wish. Sometimes even this yielding is not complete in that, for instance, the hysterical paralysis of an arm may be relieved while that of the leg may not, if the leg is more closely connected with the meaning of the forbidden impulse. However, even if all the inhibitions of the motor apparatus and of the sensory system are relieved, the forbidden wish itself may still remain completely repressed by the never really sleeping sentinel of the ego, at least through the first few hypnotic sessions and sometimes throughout a whole series of hypnotic treatments; while the ego may subsequently yield to psychotherapy in the waking state after sufficient cooperation with the therapist has been established. Even conscious material which the patient chooses to withhold may be withheld in deep hypnosis in spite of successful relief of physical symptoms by the hypnotic procedure, provided the person has a strong ego. Redlich and his coworkers⁷⁶ recently confirmed this observation also for drug-induced narcosis.

The technique of hypnosis is fairly easily learned. It consists of inducing the patient to withdraw increasingly from outside stimuli with the aid of monotonous sights or sounds. First withdrawal from sights is suggested: "You are sleeping deeply, completely at ease and relaxed; your eyelids are getting heavier and heavier so that you don't want to open them." Then withdrawal from irrelevant sounds is suggested, i.e., from all sounds other than the doctor's voice: "You are hearing only the sound of my voice, nothing but the sound of my voice." The next suggestion deals with insensibility to the ordinary varying stimuli arising in the somatic system by suggesting complete warmth and relaxation alternating with periods of extreme stiffness of all muscles. I generally begin with one arm: "Your right arm, your right hand, your right shoulder are getting heavy and warm, heavy and warm"; and then extend these suggestions over the other extremities, and finally over the whole body. Then, usually after a brief period of counting numbers, I suggest tremendous strength and stiffness of muscles for each extremity in turn, then of the whole body: "Every muscle in your body is stiffer and stronger than it ever was before; you can now hold up your arm (your leg, etc.) without any effort on your part." Then suggestions are given for the quieting of visceral sensations: "You are breathing regularly; every breath is exactly like the other without your doing anything about it; regularly and well, deeply and slowly, quite by itself." Finally a state of reduced wakefulness is reached, the somnambulant trance, in which the patient as in a dream can bring out repressed material with an ease and directness otherwise obtainable only after resistance and defenses have been overcome by months of painstaking psychotherapy in the waking state.

The physician should not be dismayed by signs of insufficient cooperation at the beginning. He must repeat his suggestions over and over again.

Once he has made up his mind that the patient will benefit by hypnosis, he must accept the challenge that he has engaged in a battle between his will and persistence to get the patient well and the patient's unconscious desire to remain sick. He must be determined that there shall be but one outcome.

If the patient's attention to surroundings or physical sensations remains too lively and the patient thus is still distractible, a good maneuver is to make the talk still more monotonous, limiting it for the time being to only one suggestion emphasized and interrupted by counting: "You will now sleep deeper and deeper—one—deeper—two—deeper—three—deeper, etc." One may then resume the other suggestions as indicated. Letting the patient wake up for a few minutes occasionally, of course on a specific signal, may likewise deepen the subsequently resumed hypnosis. This technique particularly facilitates the patient's going in deeper when immediately rehypnotized.

Flexibility and understanding regard for the particular dynamic needs of the patient are important. Thus one may frequently be able to transform an unfavorable setting or a show of negativism that may spell failure into a particularly successful treatment.

I am reminded in this connection of a 17 year old girl who was admitted to the hospital in a state of severe, apparently hysterical agitation in addition to apparent total deafness, paresis of the left arm and leg, stiffness of the left leg, and left-sided hemianesthesia. She was referred to the hospital with a diagnosis of thrombocytopenic purpura with cerebral hemorrhage, her family physician having failed to find stainable blood platelets. Neurologic examination showed normal and symmetric reflexes. Probably because of her apparent deafness the nurses on the ward felt free to discuss my treatment plans for her before she was taken to my hospital office. As she was brought in, she shrieked repeatedly, "I won't be hypnotized!" Thereupon I looked at her and said to her slowly and softly, yet firmly, "You are hypnotized already." She then dutifully and promptly obliged me by falling into a deep cataleptic trance without further ado. I proceeded to give her suggestions in a firm strong voice that she would sleep deeper and deeper, that she would hear nothing but the sound of my voice until she was wakened by the sound of a bell (kept on my desk for this purpose). I told her that she would hear my voice clearly and distinctly and that she would understand every word, that she would tell me everything that was on her mind that disturbed or threatened her, or made her feel she might lose people she loved or lose the love of these people or might have to withdraw her love from them, and that she would tell me all this because she knew that I was her physician and would help her get well and keep her well. I told her that after she had told me as much as she could think about today I would sound the bell and she would wake up, but that she would not wake up before the sound of the bell. I continued, telling her that as soon as she woke up she would be entirely well, physically and emotionally, and that then she would proceed to tell me more about her problem and we would work it out together. Her hearing promptly returned under hypnosis, and the stiffness of her left leg vanished. However, a residual weakness and hypesthesia of the left arm did not then yield to suggestion in the cataleptic stage, nor at the beginning of the somnambulant stage of the hypnosis. She then gave a weird and rambling account, which was later recognized as containing the main theme of her distressing problem in numerous disguises, as it happens in dreams. The disguises appeared so fantastic and unbelievable that her account gave the impression of paranoid schizophrenic delusions. At the same time she lapsed into psychomotor excitement in which

catatonic, choreiform, and athetoid elements commingled. When the stenographer's notes were later compared with the real story, it appeared that the psychomotor outbursts marked the times at which she came closest to giving the real story.

Because at the time I did not understand the meaning of her fantastic dreamlike allusions to events which seemed quite unlikely in reality I gradually came to the conclusion that I was dealing with a catatonic schizophrenic illness, and that the presenting hysterical features were merely incidental to an early schizophrenic psychosis. An alternate possibility not to be discounted by the negative neurologic examination was that of an acute encephalitic illness. Since I felt that I had entirely lost touch with her in the course of her cryptic, seemingly delusional talk, and her catatonoid excitement, I did not even bother to go through the motions of terminating the hypnosis, since I did not consider her hypnotized. I had her taken back to the ward and ordered an electroencephalogram to be followed by a spinal puncture the following morning. The next day as the electroencephalogram was about to be completed the warning bell sounded, indicating to the technician the approaching end of the roll of paper. The patient then woke up cheerfully smiling, alert, and to all intents and purposes completely recovered. Her hearing was restored to its former acuteness, the strength and tonus of her extremities were perfectly normal, and her sensation was unimpaired. She asked where she was, stating that she had no recollection of any events that had happened since she entered my office the evening before. She then proceeded to talk about her problem with only one more disguise, this time consciously contrived, which she gave up after further psychotherapy in the waking state. Her traumatic experience was indeed replete with the most portentous implications of danger of losing everything that had meant anything to her in her life—real, emotional, ethical, and spiritual. The pattern of her disturbance was obviously suggested by the recent illness of a close girl friend who had died from a brain tumor. The problem and her reaction to the traumatic event was worked through with her in the waking state, face to face, on an out-patient basis once weekly. In the course of several months during which her social and occupational life had resumed their normal pattern, she made a complete recovery, which has remained sustained for now ten years.

This case strikingly illustrates the remaining partial control of the ego even in hypnosis deep enough to leave the patient complete amnesia and to restore hysterically inhibited control of the motor and sensory apparatus. However, it effectively prepared the patient for acceptance of psychotherapy in the waking state. It greatly abbreviated the illness and the period of therapy necessary; this I regard as the greatest practical value of hypnosis in hysterical states. Relief of the symptom alone should never be the main or entire goal of therapy. Hypnosis should therefore be used merely as an opening phase of treatment, always to be followed by other forms of psychotherapy.

Psychoanalytic Technique

Psychoanalytic technique likewise utilizes the relaxation and withdrawal from environmental distractions incidental to the recumbent posture which plays an important role in hypnosis. It owes its origin to Freud's discovery that under such circumstances of limitation of environmental stimuli, even without the production of a sleeplike state and without direct overt participation of the therapist, but by mere voluntary cooperation of the patient, important unconscious material can get by the censor of the

ego, although more slowly than under hypnosis. In analysis the physician no longer makes the attempt to eliminate important functions of the ego such as in hypnosis, but tries to arouse the cooperation of the ego in persuading it to assume more tolerant attitudes. As emphasized in the preceding section and contrary to the standard psychoanalytic opinion, the ego is not by any means forcibly eliminated in hypnosis, since it retains important actual and potential functions even in deep hypnosis. But there is no doubt that the attitude of the physician in attempting to substitute temporarily important functions of the patient's ego in hypnosis must be a forceful one. This may explain another important difference in technique. While in hypnosis the best results can be obtained when the physician has the least previous professional contact with the patient and thus the attitude of a forceful mental overpowering is helpful in persuading the patient to allow the physician to substitute important phases of the functioning of his ego, in psychoanalysis the reverse is frequently true. It is not advisable in analysis to place the patient onto a couch immediately and expect him to produce free associations, but it establishes better cooperation if the patient is given supportive and explanatory therapy in an informal face-to-face setting beforehand. The advisability of such preliminary tension-relieving procedures has been emphasized by Knight³⁹ and Frieda Fromm-Reichmann.²⁵ For the progress of the psychoanalytic procedure, however, it is important that the analyst becomes less and less "real" for the patient (Kubie⁴⁰), withdrawing into an "analytic incognito," a development facilitated by the analyst's withdrawal outside of the patient's field of vision during the analytic hour, and by avoiding all merely personal nonanalytic communication or social contact throughout the duration of therapy.

Anna Freud²² described the position of the ego in psychoanalysis most succinctly. She stated that psychoanalytic technique requires the ego to eliminate itself, "to refrain from criticizing the associations and to disregard the claims of logical connection, which are at other times held to be legitimate. The ego is, in fact, requested to be silent and the id is invited to speak and be promised that its derivatives shall not encounter the usual difficulties if they emerge into consciousness. Of course, it is never promised that, when they make their appearance in the ego, they will attain their instinctual aim, whatever that may be. The warrant is valid only for their translation into ideas or words; it does not entitle them to take control of the motor apparatus, which is their real purpose in emerging. In deed, this apparatus is put out of action in advance by the strict rules of the analytic technique. Thus we have to play a double game with the patient's instinctual impulses, on the one hand encouraging them to express themselves, and, on the other, steadily refusing them gratification—a procedure which incidentally gives rise to one of the numerous difficulties in the handling of analytic technique."

The fundamental rule of psychoanalysis is that the patient must communicate to the doctor all his associations without modifications or inhibitions. Of course, the patient cannot live up to that rule fully nor can he make an equal attempt to do so at all times. In this situation the defensive operations of the ego come to the fore. Thus the first phase of analytic treatment is essentially an analysis of the defensive operations and the ego structure while the id itself is still protected by the defensive

operations. As the physician struggles with the defensive operations, gradually an important transformation in the doctor-patient relationship takes place which Freud²³ has described as *transference*. This means that the patient displaces, i.e., transfers, certain emotional attitudes which originally, in the dim past of the patient's childhood, had been directed toward important parental figures in relation to whom the defenses and the frustrations that caused them came first into being. Transference of such important emotional aspects of interpersonal relationships of course plays an important role in all interpersonal relationships in normal as well as in neurotic and psychotic persons. This takes place as a result of the psychodynamic law of repetition-compulsion which applies to all highly emotionally charged contents of the unconscious. But in the particularly compelling and frustrating situation implied in the gradually evolving doctor-patient relationship in psychoanalytic therapy these early emotional patterns emerge with particular vividness and force. As in the life of the child the parent is first accepted as an omnipotent protector, then doubted with disillusionment, feelings of rejection, frustration, and rage as gratification of one instinctual urge after the other has to be frustrated, postponed, or prevented, so the thwarted instinctual drives and affective needs, as they struggle to the surface again under therapy inviting the threatening risk of rejection or lack of understanding once more, bring back with them the old infantile frustration and rage.

It is this phase of the transference situation that requires the greatest degree of insight, tact, and honesty on the part of the physician. In the face of open hostility of the patient, the doctor's counter-transference is naturally not always a positive one and thus may interfere with his ability to help. The psychoanalytic training of physicians is particularly geared to enable them to stand the gaff of this battle of the transference. In general it may be stated that the physician must show an attitude opposite to that presumably shown by the important parent in the patient's childhood. By his attitude and interpretation, the physician enables the patient to work through the transference neurosis and thus to free the patient by breaking the chain of repetition-compulsion of the traumatic relationships in the patient's earlier life, thereby freeing him to form new and better interpersonal relations in his subsequent life. This working through of the transference is the most significant therapeutic contribution of psychoanalysis. As an analytic element, this procedure plays a role in other, especially eclectic types of psychotherapy as well. Every psychotherapy, however, becomes analytic in so far as it enters into this important area of interpersonal relationships. Freud²⁴ stated that the term psychoanalysis may be applied to every type of psychotherapy which recognizes the problems of transference and resistance, the basic importance of the unconscious and of the early developmental (genetic) history.

Psychotherapy Aided by Abreactive or Narcotizing Drugs

Intravenous Barbiturates (Sodium Amytal, Pentothal Sodium). *Psychophysiologic Effects.* In the early 1930's Bleckwenn,¹³ Lorenz,⁵¹ and Solomon and his coworkers⁹⁵ reported that after a period of profound sleep, produced by the injection of a barbiturate, patients who had been for months in a catatonic stupor or in deep depression began to act in a more normal fashion. In addition, it was found that there was steady improve-

ment if such treatments were repeated. Dissatisfied with the explanation that the long periods of sleep were responsible for recoveries, Lindemann^{44, 45} began experiments with sodium amytal, giving smaller amounts of the drug to both patients and normal individuals. He found that with both groups there was an increase in feelings of well being and friendliness, and a desire to communicate these feelings to others. Communication of thoughts and feelings repressed by social conventions and standard morality was released and expressed without increase in psychomotor activity. There was no change in the mental structure of the patients, but after drug administration, the abnormal thoughts, delusional ideas, and hallucinations were communicated. This revealed important material to the therapist which could be used in psychotherapeutic efforts. Thus the use of intravenous barbiturates as a means of facilitating psychotherapy by pharmacal reduction of cortical inhibitions and resistance was introduced.

During the recent war this method was particularly useful in the treatment of war neuroses. Sargant^{78, 80} introduced it during his treatment of psychiatric casualties of the Dunkirk evacuation and Grinker and Spiegel^{30, 31} studied its specific psychotherapeutic implications during the North African campaign. I had used the method before the war in neurotic anxiety tension states, catatonic stupors, and schizoaffective states when hypnosis was not feasible. During the war members of my staff, notably Captain Nicolas Camara-Peon, as well as myself, used it extensively from 1943 to 1945 for the treatment of psychiatric casualties of the Eighth Air Force and of ground combat personnel evacuated to our general hospital. Since the war I have used it with lessening frequency in civilian hospital and office practice.

The psychophysiological effect of intravenous injection of sodium amytal differs slightly from that obtained from pentothal sodium. It is important to keep in mind that sodium amytal is a hypnotic while pentothal sodium is an anesthetic. Injection of sodium amytal produces relaxation of tension at a dosage much lower than that which produces hypnosis and clouding of consciousness, while with pentothal sodium the point at which relaxation is achieved practically coincides with the point at which clouding of consciousness begins. It is for that reason probably that sodium amytal produces merely emotional and verbal expression and ventilation, while pentothal sodium produces a much more dramatic abreaction in words and actions with intense reliving of the traumatic experience. In a study which I carried out with my associates, Dr. M. R. Winston and H. Berman,³ I found that during comparable states of narcosis of apparently equal depth, the cerebral electroactivity was of higher voltage and slower frequency when pentothal sodium rather than sodium amytal was used. The question was considered whether the blocking of collaterals, which is probably the neurophysiologic correlate of narcosis, reached down to deeper levels in the brain stem with pentothal sodium than with sodium amytal. This may have some relation to the fact that the abreaction of aggressive-impulsive behavior in patients suffering from acute anxiety neuroses due to combat stress was greater under pentothal sodium narcosis (as it was under ether) than under sodium amytal narcosis in otherwise comparable conditions.

Technique. The patient should be placed on a comfortable bed in a quiet room. He should be given the explanation that he will be given an injection.

tion which will help him to relax and express himself better. He should be reassured that it is not a lie detector test and that the doctor is not trying to get anything out of him that he does not wish to tell and that the doctor is not trying to put anything over on him. He should be made to feel that the doctor is merely going to help him to be more like himself so that the doctor can understand him better. The patient should be allowed to express his objections and fears, and if he does not feel that he can rely on the doctor as his friend, he should be told that it would be better for him to subject himself to the treatment with a person in whom he can feel complete confidence.

SODIUM AMYTAL. If sodium amytal is given, a 5 per cent or 10 per cent solution should be prepared by dissolving 1 gm. of sodium amytal in 10 cc. or 20 cc. of doubly distilled water respectively. I prefer a 10 per cent solution. In most cases not more than 5 cc. of a 10 per cent solution will be required. However, the range of susceptibility to the drug varies a great deal, and it may be wisest for the first injection to have a full gram in the syringe. In preparing the proper amounts for subsequent injections it should be kept in mind that slight habituation takes place if treatments are given daily. This, however, is not invariably the case.

The injection should not be given faster than 1 cc. per minute if a 10 per cent solution is used, or 2 cc. per minute if a 5 per cent solution is used. However, it is better to give most of the injection at a rate slower than this and be guided entirely by the emotional reaction of the patient.

Usually after about 0.3 to 0.5 gm. has been given within three to six minutes the patient's tense features relax. A tear may appear and the patient will begin with very little prodding to give vent to his feelings. Depressions that have been obscured by restraint or tension will become manifest. Delusions or hallucinations that have been anxiously withheld will be verbalized. Rapport between patient and physician is intensified and the patient may accept supportive and encouraging psychotherapy as well as some tentative interpretation.

When a desirable state of ease of communication is reached, the physician should discontinue the injection and keep the needle in the vein in order to be able to resume the injection when the stream of talk slackens and blocking and tension reappear. If the patient should become sleepy, he can be brought back to adequate ventilation by waking him with an intravenous injection of amphetamine sulfate (benzedrine). It is best to have 40 mg. of benzedrine in 2 to 5 cc. solution at hand. The patient can be wakened even from deep narcosis by an injection of that amount within 20 seconds. The injection of benzedrine can be given at moderate speed; it should be stopped, of course, as soon as the patient wakes up and adequate communication is restored. The interview should be terminated with encouraging suggestions in an attitude and setting of warm emotional support. After the treatment the patient should be wrapped in warm blankets and allowed to sleep. The nurse must watch his pulse and respiration until he is fully awake and has taken a good meal.

PENTOTHAL SODIUM. If pentothal sodium is given, a 2½ per cent or 5 per cent solution should be prepared by dissolving 1 gm. of pentothal sodium in 20 or 40 cc. of doubly distilled water respectively. I prefer to use a 2½ per cent solution for this purpose of facilitation of psychotherapy because the critical dosages for facilitation of expression and for sleep are

much closer than with sodium amytal. The weaker solution therefore permits a better adjustment of the precise level of narcosis that is desirable for facilitation of expression. The rate of injection should be approximately 0.1 gm. per minute, that is, 1 cc. every 15 seconds if a 2½ per cent solution is used, or 0.5 cc. every 15 seconds if a 5 per cent solution is used, except for the first 1 or 2 cc. which should be given slightly faster. It may be wise to ask the patient to count backward from one hundred. When the count becomes slower or confused the physician should slow down the injection by interpolating a period of 30 seconds during which the injection, otherwise given at a rate of 0.1 gm. per minute, is stopped. The approach of the desired state of narcosis is signalled by a sudden change in the facial expression of the patient. Where there was a taut expression of tension before, the patient's eyes may suddenly assume a glassy stare while the rest of the face may appear relaxed. Then the stage is set for the treatment.

Unless the patient starts to communicate spontaneously, communication should be initiated and kept going by the therapist. As stated before, the element of clouding of consciousness is more marked during otherwise equivalent stages of sedation with pentothal sodium than with sodium amytal. Therefore abreaction and reliving of the traumatic situation, associated with dramatic recovery of forgotten and repressed material contained in amnesic episodes, come much more vividly to the fore with pentothal sodium than with sodium amytal. Aggressive motor outbursts are not uncommon. One of my first wartime patients suddenly jumped from the treatment bed at this moment and catapulted a typewriter from my adjoining desk against the wall missing my head by an inch. We soon learned the technique of gently turning the patient over when such behavior seemed imminent, thus letting him fight against the pillow and mattress instead of the hospital personnel. This turned out to produce equal therapeutic results with less hazard to the therapist. The above mentioned patient continued for about five such abreactive sessions to fight, tear, and bite the mattress while shouting "kill, kill, kill!" Gradually the motor abreactions became less violent and more communication came to the fore. The patient was a 19 year old bazooka man, a decorated hero who performed outstandingly for six months of fighting the harrowing actions of direct close quarters combat incidental to infiltration tactics. He had broken down after he had to kill an enemy at close range with whom he had formed a sudden bond of identification ("he was 19, just like me!"). He eventually made a full recovery.

In order to allow for the motor abreactions it is best to administer the pentothal sodium through an infusion cannula with a rubber tube of sufficient length, and to attach the syringe to the end of the tube.

Contraindications and Precautions. The contraindications and precautions are the same as those that apply for any procedure involving narcosis and anesthesia or administration of potentially toxic or allergenic substances. Oxygen, suction, a tracheal tube, and an instrument capable of pulling the tongue forward should be on hand, particularly during pentothal sodium treatment, but also during sodium amytal treatment, as well as a therapist skilled in their use. At the dosage used for psychotherapeutic narcosis, which is below the anesthetic level, accidents are extremely rare, but they may occur when least expected. Pulmonary infections, bronchitis, impaired liver function, and known drug allergies are the

main contraindications. Sudden reddening of the skin at the site of the injection shortly after injection has begun may indicate sensitivity. If this is observed, injection should be immediately discontinued.

Sufficiently large amounts of barbiturates, or smaller amounts in sensitive individuals, will produce a marked fall in blood pressure. Particular care should be taken in patients whose blood pressure is either unusually high or low and in those patients who have become weak and emaciated.

Administration of oxygen will in most cases promptly restore the blood pressure to its former level, usually within five minutes.

Another hazard is a marked loss of muscular tone with shallow respiration, which can be effectively counteracted by administration of strychnine (2 mg.) hypodermically, in addition to oxygen by inhalation.

Indications and Clinical Application. Drug-induced narcosis by means of intravenous administration of barbiturates is a method particularly useful in anxiety tension states. In hysteria, however, it is decidedly inferior to hypnosis, possibly because less active collaboration and less full acceptance of the physician as the keeper of the patient's ego is required.

A few years ago I treated a young woman who had been sick with hysterical paraplegia for thirteen years. By sodium amytal narcosis a desperate emotional outburst was produced. "I was promised I would be well by so many doctors," the girl shrieked and cried, "Oh, God must not leave me paralyzed! God will not leave me paralyzed!" But no amount of persuasion or suggestion succeeded in producing any motion of her legs. She wrung her hands, sobbing convulsively and squirming about with the upper part of her trunk and shoulders. This was repeated another time with similar results. A week later she was hypnotized without the aid of drugs; complete range of motion was restored after one hour's hypnotic session, at the end of which she was able to walk with minimal support. Strength of the legs which had been reduced by atrophy of disuse extending over thirteen years returned rapidly during the subsequent two weeks in which physiotherapy reinforced by waking suggestion was given. Occupational therapy and some rather superficial psychotherapy adapted to her limited emotional resources was given subsequently. Her recovery has remained sustained.

Even in those cases in which hysterical conversion symptoms are successfully relieved by drug-induced narcosis, the recovery remains usually less complete and less long sustained than after hypnosis. After drug-induced narcosis the hysterical patient frequently resents that "something was put over on him" that he does not understand and relapse is common. It may be that the drug-induced narcosis limits the ability of the person to understand the change. This is slightly less so in the case of pentothal sodium because it is more quickly eliminated, thus allowing a better integration of the gains achieved by the drug-induced state with the waking consciousness of the patient. This is obviously the reason why Grinker preferred pentothal sodium for his method of narcosynthesis, meaning the psychotherapeutic synthesis of the experiences undergone during narcosis.

Recently Brickner and his coworkers¹⁶ have used deep levels of sodium amytal narcosis combined with a more active form of interpretive psychotherapy which they called "direct reorientation." Their studies were based on the assumption that when the patient is in a clinical state of drug-in-

duced unconsciousness the neural machinery for the underlying fundamental patterns of behavior and reaction may be subject to reorganization. The patient comes for treatment with a written account of his dreams, and reports for a half-hour on his experiences, both inner and environmental. Then he is given sodium amytal injection. During the state of narcosis prepared scripts are read to him emphasizing important infancy and childhood situations and using the patient's own symbolic language as expressed by him either in reports of his dreams or in waking statements. Script reading—lasting for one hour unless the patient awakens sooner—is done by the staff, taking the roles of members of the family, and in some cases by members of the family themselves, which is considered of educative value for them also. Very important in designing scripts during a course of treatments are the emotional reactions of the patient during the reading period and those immediately following a session. Emotional as well as physical reactions during narcosis are recorded carefully on a special chart designed for these treatments. Script lines to which these reactions occur are underlined so that they will be noted for future reference, and important observations as to the progress of the patient can be made. Wire recordings of sessions are of great help in making accurate interpretations.

At the end of a session the patient is awakened by stirring his shoulder or lifting him, unless he has awakened spontaneously. In either event the period of awakening is an opportune occasion for intercommunication in which the patient is asked to state his thoughts or dreams. Often at this time there will be violent reactions to what has been read. Once sufficiently awakened, the patient leaves under escort.

Usually most of the intensive psychotherapeutic work is completed under narcosis, with virtually no waking therapy during the intensive treatment period. Patients frequently are not aware of their changes in attitude, or are surprised that their reactions to family and friends are different, usually attributing the change to the others. Conscious insight and interpretations are withheld from the patient until a sufficient number of the underlying patterns have been changed to a point at which the remaining work can be accomplished in waking sessions. Then the patient is given the scripts and other annotated material for study under guidance. Several weeks of intensive work are usually required for this stage. Following this there are sessions usually once a week, sometimes in a group, in which pattern changes are completed, unsolved problems are discussed, and real life situations evaluated.

Narcosynthesis treatments with or without abreaction sometimes are helpful for the relief of states of anxious inhibition which may outlast a series of otherwise successful convulsive electroshock treatments, particularly in atypical depressions. However, they are generally not as effective for this purpose as nonconvulsive electric stimulation therapy.

Sodium amytal narcosis is also being used successfully as a means to relax tension during speech reeducation therapy for the relief of aphasia caused by cerebral vascular accidents.^{9, 46, 47}

Ether Inhalation. It has long been known that the stage of anesthesia by ether is frequently preceded as well as sometimes followed by a stage of excitation. Schilder and Weissmann⁸⁵ introduced this means of modification of the emotional state into psychiatric diagnosis and therapy. The

method, however, was not widely used until Palmer^{68, 69} and Sargant and Shorvon⁸² introduced it into the treatment of war neuroses. The method produces a profound abreaction similar to that produced by pentothal sodium. In the course of this treatment once or several times the patient may suddenly become mute with a vacant facial expression, sometimes suggestive of a faint smile. Sargant and Shorvon⁸² have compared this phase to the ultraparadoxical phase of excitation described by Pavlov, and believed that the production of this phase was of critical importance for the therapeutic effect; Palmer, however, believed that the emotional expression is of greater importance.

I have found ether abreaction useful in civilian practice particularly in states of severe anxious inhibition in cases of borderline schizophrenia, and for the relief of an inert inhibited state sometimes found in schizophrenic or schizoaffective psychoses after an otherwise successful series of convulsive electroshock treatments. The use of this method in this particular connection will be discussed later (Chapter XV).

I usually carry out the treatment with the cooperation of an anesthetist. Adequate protection of the skin of the face with petrolatum ointment, and of the eyes with cotton pads is essential. The anesthetic is given slowly in order to create the greatest possible likelihood for a stage of excitation to manifest itself before the stage of anesthesia is reached. The psychotherapeutic interview is then begun. If the patient slides into deep anesthesia too quickly the cone is removed from his face and he is allowed to wake up. Excitation may then come to the fore. Anxious patients at this point not infrequently state that they felt as if they had been dying. This is then utilized for the elicitation of meaningful material in connection with that feeling, in the manner in which Frieda Fromm-Reichmann²⁵ utilizes physical sensations as clues for psychotherapeutic investigation and insight. The patient is thus helped to associate this recent vivid experience of anxiety with meaningful events in his life.

Pervitin. Pervitin (*d*-*n*-dimethyl-phenethylamine hydrochloride) has been reported by Levine, Rinkel, and Greenblatt⁴³ to be a more potent central nervous system stimulant than amphetamine and lacking the latter's side reactions. They have compared it with sodium amytal, as well as with amphetamine. The optimum dose was determined to be 20 mg. intravenously in 1.0 cc. of sterile water. Immediately following the injection the patient experienced light-headedness. Hypomanic and tense patients felt more relaxed. Generally there was increased wakefulness and elimination of fatigue; also an interesting decrease in sensitivity to pain was noted. Almost all patients showed an immediate change of behavior consisting of restlessness and over-talkativeness. There was a dramatic recall of emotionally charged material. Delusions became intensified and florid fantasies were brought to the surface and elaborated. Nonpsychotic patients experienced a general feeling of relaxation with euphoria, relief of tension, and increased alertness. Forgotten memories and traumatic experiences were recalled. The neurotic patients displayed evidence of aggression and confident self-assertiveness. The psychopathic patients talked freely, without covering up. Two hysterical patients responded dramatically to pervitin. As to side effects, the patients usually complained initially of dryness of the mouth, thirst, tightness in the chest, and difficulty in breathing; loss of appetite usually persisted for 24 hours.

Pervitin evokes a more spontaneous, richer, and more appropriate re-

sponse than sodium amytal, and is sometimes effective when sodium amytal is not. There is no loss of consciousness or amnesia, and all material dealt with in the pervitin interview may be recalled. Amphetamine has similar physiologic effects, but less intense psychologic effects. Pervitin may counteract the hypnotic effect of amytal, and amytal may calm the overexcitement occasionally induced by pervitin.

Carbon Dioxide. After the original discovery of its influence on abnormal mental states by Loevenhart, Lorenz, and Waters⁴⁹ and by Solomon and his coworkers,⁹⁵ carbon dioxide was introduced into psychiatric therapy by Meduna.⁵⁶

The treatment is best initiated with 25 per cent carbon dioxide in oxygen, to be gradually replaced by pure carbon dioxide as soon as the patient is relaxed and breathing deeply. The patient is asked to raise his hand while the doctor counts each breath with a loud voice. After about twenty breaths of pure carbon dioxide the patient stops raising his hand and becomes unconscious. He may stiffen out momentarily and make a loud outcry. A brief period of motor excitement with thrashing about of the arms and legs is followed by limpness and rapid return of consciousness. The patient then remembers and is able to report a vivid dream, usually containing an experience of immense brightness of light and vividness of colors. The dreams may be expansive and in keeping with power drives and are usually replete with wish fantasies. They are seldom frightening.

Wilcox¹⁰³ has paid particular attention to the meaningful aspects of the carbon dioxide dreams and has utilized them in subsequent psychotherapy. Wilcox has also used the carbon dioxide treatment as a test of the rigidity of the patient's defensive operations by subjecting the patient to questions aimed at a probing of his defenses before and after treatment ("psychopenetration"). Meduna⁵⁶ and Wilcox¹⁰³ considered carbon dioxide treatment helpful in neurotic anxiety states and in alcoholism. Whether or not the physical aspects of carbon dioxide treatment play the leading role in their unquestioned results remains open for further investigation. I have recently obtained evidence that the psychosomatic effect of the treatment is sometimes similar to that of nonconvulsive electric stimulation therapy and of insulin subcoma treatment, in that it induces a downward shift on the psychophysiologic graph, sometimes to that of combined convulsive-nonconvulsive treatment, in that it reduces the response in either direction (Chapter XIII, Fig. 63). We are justified in concluding that carbon dioxide represents a facilitating agent—physical and situational—in the hands of some therapists. I have found it a helpful adjunct to the psychotherapy of schizoaffective and schizophrenic borderline states, particularly in those complicated by pseudo-psychopathic acting-out behavior.

Psychotherapy by Personal Interview

Modern psychotherapy has incorporated as many practical features of the above described special techniques as feasible, in addition to its basic ingredient which is solid emotional support through interpersonal communication. This latter and most important phase of psychotherapy is best accomplished by the personal interview ("face to face") technique. A great many useful contributions to the principles and techniques of psychotherapy have appeared, notably those by Whitehorn^{101, 102} and by Fine-

singer.²¹ Also Frieda Fromm-Reichmann²⁵ in her splendid volume, *Principles of Intensive Psychotherapy*, appears to have relaxed the rigid limits of classical psychoanalytical practice by using the face to face technique as well as a large measure of supportive therapy.

In an earlier chapter (VI) I have attempted to develop for the reader the general principles of psychotherapy from the supportive, through the ventilative and abreactive, to the interpretive procedures. It is the goal of eclectic psychotherapy to use all helpful measures to best advantage.

The supportive part of the treatment program should be initiated by thorough attention to the patient's complaint and problem, best accomplished by interested listening.

The patient's communications may be gradually steered into ventilation and abreaction which can be accomplished in the proper setting—and with the right emotional stimuli and cues—without the use of drugs. Experience with the techniques of drug-induced abreaction is of course helpful to the therapist in his well planned efforts to guide the patient into giving true vent to his feelings. Among the interpersonal influencing factors which he must use, and by which he can help to bring about ventilation and abreaction, are permissiveness, understanding, and respect for the patient's feelings.

The stage of interpretation should be approached with restraint and consideration best described by what I have called earlier (Chapter VI) a shift of emphasis. Supportive measures—after understanding and rapport have been established—should now also include persuasion, suggestion, and reassurance; these should be continued throughout the interpretive phase of the treatment. The interpretive phase itself is of course of paramount importance.

In giving interpretations a carefully planned approach—consisting of four distinct phases—should be carried out. These should be adapted to the particular defensive operations of the patient with which the physician by that time should have become familiar. These four phases are: (1) to offer the patient tentative insight that certain of his actions and reactions are not understandable on logical grounds alone; (2) to make the patient understand that these actions or reactions may be due to some highly personal, yet not uncommon experiences that have happened to him and therefore may be understandable as a result of a fairly compelling emotional reaction that has its deep unconscious meaning; (3) to make clear to the patient that the logically un-understandable action actually accomplishes a disguised satisfaction of the deep unconscious needs aroused by what has happened to him; (4) to help the patient to find better ways for the satisfaction of, or better defenses against, these unconscious needs.

I should like to give a simple example. If one wants to explain to a person that the reason why he and other people avidly devour the gory murder stories splashed on the front pages of newspapers is that they harbor an unconscious hostile urge of their own, there are two ways of telling it to the average person. One way would be to tell him, "You are not really any better than that murderer. You want to murder just as much as that man. The only thing that is different about you is that in addition to being a potential murderer you are also a Caspar Milquetoast and a coward." That certainly would get the average man's back up and he

would emphatically defend himself against this accusation, which really is an overstatement of the truth if put that way.

The other way to tell it is as follows: (1) You mention to the man, "Isn't it strange that newspapers talk a lot about the murders of the day, and not about the many more people who live happily? Why is it that the newspapers play up these gruesome accounts of murders to such an extent, and that the average man laps them up so eagerly? There are certainly more pleasant, important and constructive news items than those to print. Why is it that stories of the opening of a new school building, or the celebration of a golden wedding—news stories pleasant to contemplate from the point of view of the progress and happiness of mankind—are relegated to the inner pages or omitted entirely?" Your listener would readily admit that this is certainly a strange fact.

(2) The second step would be to tell the man that there may be a highly personal reason slightly different for each person, but basically similar, which is so highly personal that most people would not be consciously aware of it. This reason is that most of us are subject to a great deal more frustration than we are willing to admit to ourselves. It is a rough life and we have to put up with a lot. There are many people who try to take advantage of us and we get little appreciation and understanding, frequently not even from our immediate families. All too often we feel friendless, forsaken, our intentions misunderstood, rejected and crowded to the wall. No wonder then that every one of us may sometimes feel like killing someone, though of course we don't do it. That is true enough, the man in the street may say to that argument.

(3) The third step is then to establish the link between the repressed aggressive feeling explained in step 2, and the otherwise not satisfactorily explained fact advanced in step 1. This link may be established some way as follows: "Well, this being so, maybe we get something out of our systems by following so avidly those murder stories." The average man may concede without too much emotional protest that this could quite well be so.

(4) The next and fourth step is then to point out that there are other and better ways to get repressed anger, rage, and frustration out of one's system. One could work twice as hard "just to show them," or one could hit golf or tennis balls instead of thinking of hitting noses; or one could dig oneself into a job instead of indulging in some fantasy of personal aggression or in the unconscious vicarious gratification afforded by aggressive acts committed by others.

This is merely an abstract theoretical example of the schema of the steps to be taken, but they apply, of course—sometimes slightly less clear-cut—to more practical situations to be dealt with in everyday psychotherapy. I should like to give an example from a recent practical experience.

A widow in her sixties developed a severe depression in a setting of increasing rejection and dwindling of narcissistic supplies from her only son. This state of affairs gained momentum in a vicious cycle pattern by her unfortunate and compulsive scrutiny and criticism of all the expenses or purchases incurred by her son on behalf of his wife and home, in spite of the fact that her son most dutifully took care of all her needs and comforts as well,

which she herself admitted reluctantly. After her recovery with the aid of convulsive electroshock treatment she was quite ready to admit there was no need for her constant interference in and continuous uncalled for debating of her son's budget, since he was quite capable of managing his own affairs as well as caring for her welfare, and she was willing to accept that her preoccupation had a reason which was not quite logically explainable. It was then pointed out to her that this preoccupation could be due to some frustrating personal experiences that had happened to her in this particular area of relationship in her own marriage. With little prodding a tale of woe then spurted out about how she had slaved and skimped for her husband, a taciturn, unaffectionate, and miserly man who took everything she did for him and their son for granted. If she got a new hat every eight years it was a good deal and she literally had to fight for every new dress she was allowed to buy. He never understood that anyone would want to spend money for new clothes or for entertainment, such as the theater.

This ventilation of her old resentments led easily into step 3: the establishment of the link between her frustration and thwarted needs and her otherwise inexplicable present attitudes. She readily admitted that strong feelings erupted when she saw her daughter-in-law get everything that she had always wanted and never got; although she was still for a while hiding behind the rationalization that it might not be good for her daughter-in-law to get that much. However, she abandoned this position when she was made to understand what a happy woman she would have been if she had been married to a man as well adjusted and understanding as her fine son.

Step 4 then followed and consisted in changing her poor defense—displacement of her frustration and hostility engendered in her relations with her late husband—into a better defense, namely, identification with the young couple. This was done by giving her strong support by pointing out that after all it was her son, and that it was the love and the upbringing that he had received from her that made him capable now of giving love and understanding to his wife and to honor his wife as if she were his mother. Thus the patient was enabled to identify herself not only with her son as her own flesh and blood and the product of her education, but also with her daughter-in-law as a symbolic extension of herself. Her late husband and the memories of her battles with him were thoroughly buried, enabling her to become free from the repetition-compulsion of her pattern of nagging ineffectual rage. Her poor defense of displacement was changed to the normal defense of identification.

Involvement in a direct transference situation was avoided with this woman except of course as implied by her constructive rapport with the "good," permissive, understanding therapist who provided the new life experience that was instrumental in releasing her from the negative, frustrating hostile relationship with her late husband. The reestablishment of harmonious relationships with her son and daughter-in-law has so far remained sustained.

It is sometimes possible to carry out this type of rapid psychotherapy in certain psychotics, particularly paranoids, without the aid of previous physical treatment. I treated such a patient in 1948.

An insidious and severe paranoid development had been set off five years earlier by the untimely death of his favorite son at age 8 from rheumatic fever. As these years progressed he continued to accuse his wife of having caused the death of the son by her negligence although the wife, a devoted and attentive mother with four other children both younger and older than the deceased, was almost the paragon of an affectionate, attentive, and resourceful mother. Not only did he withdraw his affection from his wife but also

from the other children as well. The only boy that had mattered was the dead boy, and now everything—home, children and marriage—was spoiled. By scurrilous hostile remarks, rejecting reductive attitudes and by deliberate and demonstrative absences from the family table (he preferred to gulp his food alone in a corner in the kitchen or cellar, refusing to join the family at the table—"for me eating is just refueling"), he made life miserable for his wife and his four children. There was also deep resentment against the township in which he lived, and when one of the town officials died, he believed triumphantly that he had killed him by the concentration of his hatred. All that remained of his capacity for positive cathexis went into his work and into athletic activities, and interestingly enough, to the boys at an orphanage and to the inmates of a reformatory for boys and adolescents whom he gave what free time he had in teaching night school. A lively island of feelings of sympathy for underdogs and those who were wronged by life emerged as a remaining positive asset.

It turned out—as he recounted the moving and in many ways tragic history of his life—that he had lost his father at an early age. After a few years of trying to make a go of it, his mother, left widowed with six children, signed him away to an orphanage famous for its harsh discipline, while keeping two of the younger children with her. After graduation he was discharged into the custody of a farmer, then worked his way through high school and university, finally arranging for his mother to come and live with him, only to be disappointed by her lack of affection. In one way or another, he had been at war with the world ever since, although his marriage as well as the relationship with the children had originally been warm and satisfying until the tragic loss of the second and favorite boy.

In a total time of three continuous hours I succeeded, using insight therapy as well as techniques akin to religious conversion, in changing displacement into identification. The patient was told, "You have been hurt a good deal in your life. You were hurt first by your father dying when you were a little child, then by your mother in that she showed no sympathy and understanding for what you had been through when she returned to make her home with you, and that she did not give you the warm affection you needed. And you were hurt again after that by the various things that have happened in your life. It is for that reason that you have built a shell around yourself in order not to get hurt anymore. But that is what keeps the warmth and affection that your wife and family need all bottled up within you, and the only people it can go out to are the little boys at the orphanage and the young men at the reformatory. But now, there is no more need for that shell. You have succeeded. You are a popular figure, highly respected, and you have achieved material success and security. You can really let the defenses down now. Nobody is going to hurt you anymore, except you yourself, by not living in the atmosphere of warmth and love which you need. Your wife really loves you. She loves you deeply and you know it. And you both will be much happier if you can give her the affection and warmth she needs." The result was amazing. The patient came back two weeks later to all intents and purposes cured. "My wife certainly is making a great effort," he declared. "It is very pleasing, and I'll certainly cooperate to see whether we get along without a few fights for a while!" His wife wrote me enthusiastically six weeks later: "My husband is very much improved. It becomes more and more apparent." Two months later she became more outspoken, saying, "what you have done for us is really a miracle. It becomes more so all the time. One day our daughter said, 'We are getting back to the old times. Daddy isn't mad any more. I haven't heard him yell for a long time.' I am getting over being tired and inefficient and lazy, and life seems good again. My husband is relaxed and calm and seems much happier. The same things that used to irritate him

still occur at times, but they don't bother him any more, at least not much." And still later she wrote, "The magic is still working. It's quite unbelievable, but there it is, so I can no longer be skeptical about it."

In this case also, a fundamental change in the defensive position of the patient was accomplished by changing displacement and projection into identification, without any significant utilization of the transference relationship with the physician himself.

Of course the development of a transference neurosis is sometimes necessary and inevitable, particularly in certain schizophrenic borderline states and certain neurotic anxiety tension states which would otherwise remain resistant to psychotherapy. In those cases the transference relationship has to be worked through in the manner developed by the psychoanalytic school, as briefly summarized in the section on psychoanalytic technique (see page 292).

*Psychotherapy by Conference, with Specific Reference to the
Psychotherapy of Married Couples*

Contrary to the opinion held by many psychiatrists that the same therapist should not treat both partners of a marriage if they both happen to have mental or emotional problems, I believe that it is not only feasible but particularly beneficial and effective if the psychotherapist is able to take on both the husband and the wife as his patients. This point of view is held by most marriage therapists, including Laidlaw,⁴¹ Allen and Appel,⁴ and Mudd.⁶⁵

In order to do so the therapist must of course be completely sure of his objectivity and impartiality; otherwise he could not give adequate support and permissive and approving understanding to the parties at war, whether it be a "cold war" or an overt one. If the psychiatrist should find that because of his own countertransference in favor of the less guilty party he may be punitive or unsympathetic toward the guiltier one, he should, of course, refer one of them to a colleague. This, however, will only rarely have to be the case.

The understanding of the issues at stake will frequently be greatly aided by a three-cornered interview either at the beginning of therapy or sometimes after some understanding of each of the partners has been gained by a number of preceding individual interviews. The late Dr. Abraham Myerson⁶⁷ who advocated this procedure used to call it "a free-for-all with an umpire present." This type of interview is an excellent means of reality testing as well as a means of gaining insights into the defensive maneuvers and the pretenses of the two main parties.

After further progress in individual psychotherapy has been made these interviews should be repeated, and should then become an excellent means of working through specific issues as well as of giving an opportunity to each of the two marriage partners to gain insight into the emotional needs of the other. It also makes it possible for each partner to allow and invite insight into his own emotional needs by the other. The topics for these subsequent three-cornered sessions should be well prepared and gone over with each partner beforehand so that no material will be revealed or brought into the open concerning each of them without his or her previous permission. It is essential that both partners must have full conviction that

the psychiatrist will not reveal any confidences without full previous approval.

The most important insights derived from these confrontations are concerned with the realization of the repressed and thwarted needs for affection which frequently underlie bullying, overbearing attitudes on the part of the male, or nagging, demanding aggressiveness on the part of the female. These problems will of course have to be patiently worked out first in individual psychotherapy, the joint interviews then serving as a means of reality testing. Thus the psychotherapist may play Shakespeare's Prince of Aragon to many a long married Benedick and Beatrice. He must be careful, however, not to play the overforceful role of Wagner's Siegfried to many a mismatched Gunther and Brunhild, except possibly by merely the remotest implication of identification.

In this connection it may be useful to clarify in our minds the main obstacles to a congenial marriage as well as the main positive supports of marriage from the psychiatrist's point of view. The causes of failure in marriage are both general and specific. One of the most important general causes is an unconscious rebellion against the cultural restraints imposed by the institution of marriage itself. The specific causes could be defined as the many possible difficulties arising out of the relationship of two specific people.

What makes marriage successful and holds it together, apart from the restraints of temporal and spiritual law, consists essentially of four solid foundations, three of which are the minimal essential requirements for an inherently stable marriage: (1) biologic and physical compatibility: that is, the ability of the marriage partners to give each other solid physical satisfactions; (2) mental compatibility, congeniality, and mutual understanding; (3) love in the social and spiritual sense of the word; (4) economic security. If three of these factors are present, the marriage has a good chance of succeeding even without a fourth. But I feel that the psychiatrist can help a couple iron out almost any number of difficulties if the biologic foundation of their marriage is satisfactory. At the base of many unhappy marriages is the lack of solid sexual satisfaction, other difficulties merely indicating surface disturbances arising from sexual maladjustment.

It is an important feature of therapy to bring this out into the open and to treat sexual disturbances effectively, with the aid of medical measures if necessary, as well as by psychotherapy. In the case of frigidity in women it is sometimes useful to enlist the aid of a well adjusted married woman, either a woman doctor or a social worker, to treat the patient because a therapist of the same sex may utilize to the hilt the therapeutic effect of identification and mimesis, thus accomplishing what the patient's mother failed to do for her. The elements of identification and mimesis are also of importance in the group therapy of frigid women to which reference will be made in the next section.

Other sources of difficulties in marriage arise in the sphere of personal congeniality. Most disturbances here are caused by unrealistic demands or neurotic attitudes. Many persons marry a social stereotype and expect social stereotype responses, only to find that they are dealing with a unique human being whose responses they had not anticipated and cannot handle.

Group Psychotherapy

Group psychotherapy is inherent in many ritualistic practices of the past and present, which undoubtedly had and have an incidental group-psychotherapeutic effect (Sargant⁸⁴). Group psychotherapy planned under medical auspices had its origin in the work of Pratt at the Boston Dispensary,⁷⁰⁻⁷⁴ who in 1905 founded a group clinic for patients suffering from tuberculosis. Later he extended this approach to the management of other illnesses as well; in 1930 he founded a group clinic for psychoneurotic patients called the "Thought Control Class," subsequently renamed "Classes in applied psychology". The mental hygiene movement as well as the society of "Alcoholics Anonymous"—which will be discussed at greater length in the chapter on alcoholism—likewise blended medical, hygienic, social, inspirational, ethical, and religious ideas. Specifically psychiatric group therapy was initiated in 1921 by Lazell⁴² for the treatment of schizophrenics, and extended to broader groups of psychiatric illnesses by Schilder.⁸⁷

Psychiatric group therapy received its greatest impetus during the recent war. The main reason for my use of it was as a means of saving time in the course of psychotherapy which I regarded as essential for all of my patients. It began in the form of orientation lectures preparatory to individual insight therapy. Patients who had done well on various forms of other therapy, particularly somatic therapy such as deep sleep or insulin subcoma, combined with supportive psychotherapy, were obviously in need of an explanation of their illness, the meaning of their improvement or recovery, and some information about their chances for health and performance in the future. In order to save time and multiplicity of effort which would have been necessary for each patient to receive these explanations individually, patients whose personalities, educational and military status, treatment results, adaptation, and progress were considered similar, were collected in a group and given a series of lectures. These began with an exposition of the Cannon-Lange theory of neurosis as stress reaction, followed by a lecture on the way in which stress reactions tied in with earlier personal experiences, followed by a third lecture in which healthier patterns of adaptation were suggested. Particularly important were explanations aimed at restoring the patient's self-respect. The answer to the question, "What happened to me, doc? Was I yellow or something?" had to be given in a helpful and constructive way facilitating the acceptance of insight. An explanation of the means that existed to increase stress resistance, and of the adoption of better defensive operations on the basis of better insight into instinctual needs had to be taught to some groups; acceptance of limitations of stress resistance plus maintenance of the desire to keep up performance with self-respect, such as needed for reassignments to noncombat duty, had to be taught to others. The latter group was aided by arousing identification with the therapist and other hospital personnel who likewise served in a useful yet noncombatant capacity.

In the process of this work it was quite evident to all who took part in it that very soon what had started as a lecture with question-and-answer period between patient and doctor had become a means of exchange of ideas, feelings, and emotions among the patients as well. Insight was aided by understanding of the problem of the other fellow, which always comes easier than self-understanding. Leaders emerged; identifications and other

interpersonal reactions became obvious. "If that fellow can go back to combat duty, I certainly can! He was much sicker than I ever was." The therapeutic process frequently progressed without any further active push on the part of the physician acting as group leader. Gradually new concepts of group interaction emerged and the simple processes of the lecture and meeting evolved into a complicated psychotherapeutic process with definite dynamics of its own.

One principle, however, soon became obvious to me: that all patients in group therapy have to have simultaneous and continuous access to individual psychotherapy as well. This aspect tends to become all too often neglected with the increased routinization of the procedure, which is thus in danger of becoming a devitalized and ineffective fad, although the pioneers of group therapy, especially Slavson,⁹³ have pointed out this basic need emphatically: "By and large, it is an error to speak of 'the group' as an entity in therapy. It is always the individual, and not the group as such, that remains the center of the therapist's attention. The group is merely a means for activating individuals and supplying the kind of experience that helps modify feelings and attitudes. . . . To consider the group as a *treatment focus* instead of as a *treatment tool* misleads us as to the aim and means of therapy. In this connection, it may be observed again that there is a large number of patients who should not be placed in groups because of the harm that may result from multiple relationships and tensions."

There must be detailed planning of the group before it can function properly as a psychotherapeutic tool. The membership must be limited in order to achieve a familial type of relationship. Cultural and educational backgrounds of the members must be similar in order to foster the conversational rapport. The group should not be a chance conglomerate of persons who happen to have the same real, emotional, or personality difficulties. The individuals who comprise the group should show a need for recognition, a desire to belong to a group, "social hunger."

Slavson⁹³ has pointed out that while the psychopath would seem to be a good subject for group therapy because of his regular attendance and obvious satisfaction in group activity, such is not really the case. The psychopath needs the protection of a less personal involvement. He cannot utilize the freedom of group therapy, but needs a more authoritarian framework of reference in the psychotherapeutic relationship. It is rather the psychoneurotic who benefits most from group therapy. His "social hunger" is the direct result of rejection patterns formed in childhood.

The groups may be activity groups or interview groups. Activity groups are particularly appropriate for children, especially those under 13 years of age. Children likely to benefit are those neurotic youngsters or children with neurotic traits who have inadequate social contacts, are unable to get along with other children, or have a need to express aggression. In interview groups, psychotherapy is essentially of the same nature as in individual psychotherapy, but the presence of others with similar problems gives each member support to reveal his feelings and attitudes (Slavson⁹³). The basic elements are: catharsis, insight and/or ego strengthening, the working through of transference relationships, reality testing, and sublimation.

Greene²⁹ has contributed some very practical rules for the planning

and organization of therapy groups. Among other points he emphasizes: (1) The therapist should not show discrimination among patients—he may call attention to shortcomings and unhealthy personality traits and reactions, but at no stage should he withdraw approval from the person himself. (2) The therapist should never force patients into a situation in which they may feel themselves in an inferior position. (3) Patients in any one group should present problems that are essentially similar and should be in the same general phase of treatment. (4) Patients should be exposed to different therapists at different times. (5) The therapist should be acquainted with the individual patient's history and background. (6) The basic goal of therapy should be restated frequently. (7) The therapist should be easily available for private discussion of individual problems. (8) The group should meet frequently, if possible several times a week. (9) There should be a variety of supportive group activities to supplement the actual therapeutic sessions. This requirement was recognized early by Merrill Moore and his co-workers,^{34, 57, 61} who have developed an effective combination of group therapy with diversional, recreational, and occupational therapy in their treatment of psychiatric war casualties.

As to special application of group therapy, Geller²⁶ finds best results with alcoholics and neurotics. Wender¹⁰⁰ found group therapy of value on a ward of insulin-treated patients in combating feelings of loneliness. Among nonpsychotic adults, Sheldon and Landsman⁹² had results in college groups with academic difficulties; reading skill remained the same but personality factors improved. Van Emde Boas⁹⁹ reported results with anorgastic women—improvement was reported after three, seven, thirteen, nineteen, and twenty-eight sessions respectively. Stone and Levine⁹⁷ had good results with sexually maladjusted couples; sessions were held separately for husbands and wives. Lloyd⁴⁸ reported good results with mothers of children who presented various problems, Bixby and McCorkle¹¹ with prisoners, Randall and Rogers⁷⁵ with epileptics, Slavson⁹⁴ with delinquent children and adolescents. Day, Day, and Herrmann¹⁷ employed the method as a means of alleviating the build-up of anxiety and development of regressive patterns of defense encountered in patients suffering from chronic debilitating organic disease of the nervous system, such as multiple sclerosis.

Semrad and his co-workers,^{20, 27, 32, 53, 54, 89, 90, 91, 96} encouraged by the clinical observation that ward patients who experienced a communal social life improved more rapidly than private-room patients (see also Bockoven and Solomon¹⁴), undertook the important and difficult task of developing practical and effective methods of group therapy adapted to the needs of hospitalized psychotic patients. Their methods are still in the process of evolving, and their results in the process of evaluation; important steps in the right direction, however, have been accomplished; among them the working out of methods of resolving anxiety among therapists engaged in this type of treatment. As to the progress of therapy, Mann and Semrad⁵⁴ distinguish four stages: "Stage one is a testing situation that culminates in group identification and unity. In stage two there is free expression of anxiety-laden psychotic material with attempts on the part of the patient to explain such mechanisms. Stage three is one of introspection, mutual criticism, and working through of emotional problems. Stage

four is not entirely clear but does seem to be concerned with discussion of future plans on a reality level.

"Therapeutic results in terms of benefit to the patient, to the hospital, and to the therapist are quite specific. The self-imposed isolation of the patient can be broken and interpersonal relationships re-created. In respect to management, untidiness and assaultiveness are diminished, while a general atmosphere of helpfulness permeates hospital personnel and is expressed in improved attitudes of such personnel toward the patients. The therapist learns with the group and finds that the group contributes to his own emotional growth."

A special variant of group therapy, combining elements of activity as well as ventilation, abreaction, and insight therapy particularly adapted to the means of expression and understanding of the psychotic patient, is the psychodrama, developed by Moreno.^{63, 64}

Occupational, Diversional and Recreational Therapy

Occupational therapy is one of the most potent auxiliary forces in the rehabilitation of patients suffering from mental disease. Merrill Moore,⁶⁷ one of the most persistent practical advocates of occupational, diversional, and recreational therapy for hospitalized as well as nonhospitalized patients, states: "I have seen psychotic patients make a recovery largely through the doorway of occupational therapy and the psychotherapy that goes with it, group and individual." Moore feels that occupational therapy should begin early in the form of participation in the routine activity of ordinary duties about the ward. He found that mere participation in ward duties is already instrumental in relieving the characteristic feelings of guilt over being sick and of anxiety over being disabled which many patients experience upon entering a hospital. "If the nurse gives the patient something to do, something the patient can be reasonably expected to accomplish or to complete, and if the patient does it, he receives approval from the nurse and commendation from the doctor; in fact, the whole structure of the illness situation becomes tinged with positive values that otherwise might not be present."

There is an element of transference in that situation: it is as if the doctor were the father, the nurse the mother, the patient the child, and the hospital the home.

The next step in occupational therapy should be the cultivation of more intricate specific tasks or interesting and absorbing hobbies. The choice of the proper tasks or hobbies for patients or groups of patients is of greatest importance. Barton,^{7, 8} who during the war was in charge of occupational therapy in the Army's chief psychiatric consultant's office, made an important contribution in alerting occupational therapists to the need for a varied as well as meaningfully significant program. In all too many places, occupational therapy had become synonymous with weaving and basketry. Barton referred to the jibing headline in a San Francisco newspaper during the war which proclaimed: "Marines Take Up Knitting!" although of course he conceded the point that anyone would rather cut out paper dolls than look at the cracks in the ceiling all day. Largely as a result of the stimulation from the psychiatric consultant's office, meaningless activities, or activities adversely meaningful in that they decreased rather than

increased self-respect, were soon replaced by adult and stimulating projects among which the carpenter's shop, the machine shop, and the armorer's shop soon became the most popular. Barton⁷ also suggested that all the varieties of occupations that an occupational therapy department might offer should be listed and presented to the patient so that he could check the ones he thought he would like most, as well as the ones he thought he would like least.

Merrill Moore⁵⁷ suggested creative writing and literary clubs as particularly stimulating projects for patients with intellectual aspirations. Blackman¹² used the issuing of a ward newspaper to good advantage. Among absorbing hobbies Merrill Moore^{58, 59, 60} found the collection of sea shells particularly interesting. He also used this type of project conveniently during his work with neuropsychiatric casualties on Pacific islands during the war. Barton⁸ suggested that jobs should be graded from simple ones, like raking leaves, to those of considerable complexity, like assisting the treasurer in making his reports. He observed that paranoid patients with delusions may sometimes be helped by a feeling of importance in their work, accomplished by giving them a job in the hospital which develops their sense of importance in a constructive way. In some instances remarkable results have been achieved by merely putting the patients together to work as a team. In this way a sense of awareness of others has been established, which is the first step toward becoming a part of a social group. Chronic patients profit from an activities program which leaves them little time to ruminate on delusional ideas.

Moore⁵⁷ considers occupational therapy as part of Myerson's⁶⁶ "total push" method of treating mentally ill people. He feels that all activities that give them outlet or stimulation or assist them in abreaction, or transference reactions, or new object relationships (object cathexis), or the formation of new patterns and techniques of doing things could be valuable in helping them distribute their libido or modify their pattern of energy distribution in ways that are favorable to the individual and the institution—and the group to which the individual belongs. Barton⁸ is careful to point out that while hospital industry can thus be a great resource in the resocialization of patients, it can never replace or compete with industrial schools or vocational training centers, for which there is great need after the discharge of the patients from the hospital, especially those of the younger age group.

Supplemental Medication

The most important use of medication in the treatment of emotional and mental disorders is probably for the restoration of sleep (Myerson¹¹¹). Medication for restoration of sleep is frequently unnecessary when physical treatment, such as electric shock, is used, because one of the earliest therapeutic benefits frequently derived from electric shock is restoration of the patient's ability to sleep.

The choice of the most appropriate nocturnal narcotic depends somewhat on the type of sleep disturbance, whether it is difficulty in falling asleep, frequent waking at night, or early waking in the morning. When all three of these disturbances are present the best nocturnal narcotic is sodium amytal, 3 to 6 grains. The dose depends in part on the weight of the patient and on the severity of the emotional tension that prevents

sleep. It is of paramount importance to supply a sufficient dose since the amount required by emotionally disturbed persons is much greater than that required for the incidental sleep disturbances of those who do not suffer from gross emotional or mental disorders. If the sleep disturbance is merely one of the inability to fall asleep, the new dormison (Schering) is helpful, the additional advantage of this drug being that it produces no hang-over the next morning. Two capsules before bedtime is an appropriate average dose.

Medication designed to relieve depression and anxiety during the day can be effectively designed on the basis of the recognition of the reciprocal pharmacologic effects of amphetamine (Benzedrine) sulfate and the barbiturates (Myerson¹¹⁰). Amphetamine and its derivatives, such as Dexedrine, relieve depression but may enhance anxiety. The barbiturates, on the other hand, may relieve anxiety but may enhance depression. Useful dosages are 5 to 10 mg. of benzedrine or dexedrine morning and noontime, after eating. Amphetamine and its derivatives should not be prescribed to be taken during the evening because they interfere with sleep. Of the barbiturates, recommended dosages are: phenobarbital, $\frac{1}{4}$ to 1 grain, three times a day; sodium amytal, $\frac{1}{2}$ grain, three times a day; sodium butisol, $\frac{1}{2}$ to $1\frac{1}{2}$ grains, three times daily; or mebaral, $1\frac{1}{2}$ grains, three times daily, orally.

Myerson¹¹⁰ first discovered that combinations of amphetamine derivatives with a barbiturate may have a more balanced effect than the use of either drug alone, in that their combination may produce tranquillity instead of merely either sedation or stimulation. Suitable combinations, to be given in the morning and at noon, are: benzebar, which contains $\frac{1}{13}$ grain (5 mg.) of Benzedrine sulfate and $\frac{1}{4}$ grain of phenobarbital; dexamyl, which contains 5 mg. of Dexedrine sulfate and $\frac{1}{2}$ grain of sodium amytal; and syntil, which contains $\frac{1}{4}$ grain of butisol sodium and 2.5 mg. of syndrox hydrochloride (methamphetamine hydrochloride).

Tolserol, a curare derivative, relieves anxiety and lessens manic drive without other effects of sedation, such as slowing of thought processes or sleepiness. Recommended dosage is 250–500 mg. four times a day, orally.

Hormone Treatment. The first well controlled study of the use of hormones in the treatment of depressions was that of Sack,¹¹⁶ who used both estrogen and progesterone (corpus luteum hormone). By and large the results of hormone treatment in depressions were disappointing for, in spite of the popular belief that late or middle life depressions in women are related to the menopause, this is not so in most cases. However, although they are rare, true menopausal depressions do exist and they of course respond superbly to a well planned estrogen and/or progesterone therapy. It is wise to plan the treatment in collaboration with an endocrinologist or gynecologist interested in endocrinology. In regard to their autonomic response pattern, true menopausal depressions are usually characterized by marked epinephrine-precipitable anxiety as well as by a paradoxical reaction to mecholyl, in that the blood pressure rises after intramuscular injection of 10 mg. of mecholyl, sometimes after a very brief drop. It is important, however, not to pursue hormone treatment for too long unless results are forthcoming after the first ten doses which may be administered daily. If no therapeutic results at all are obtained by that time, further treatment will be ineffective and other appropriate forms of physical

treatment should be substituted. Conversely, if other forms of physical treatment remain ineffective in women in the menopausal age group, or produce only partial improvement, the addition of as little as 2 mg. of progesterone (given intramuscularly twice monthly) on the twelfth and fourteenth days following the first day of the preceding menstrual cycle, or at the approximately equivalent time in amenorrheic patients, may bring about recovery (Billig and Bradley¹⁰⁸). These authors found that injection of 2 to 5 mg. of progesterone (three injections per month) was helpful in puerperal psychoses and recommended that this medication be continued for ten to twelve months after recovery. They regarded disturbed estrogen-progesterone balance as an etiologic factor in the onset of these psychoses as well as in their reactivation or relapse. Premenstrual onset or exacerbation, as well as onset during puerperium—the two periods having rather similar steroid metabolism—is diagnostic indication of this type of triggering of the mental disturbance. The authors concluded that decreased progesterone production in these cases leads to relatively excessive or unantagonized estrogen production contributing to irregularities of the autonomic nervous system.

Altschule and Tillotson¹⁰⁴ introduced testosterone as an agent in hormone therapy of depressions, in males as well as in females. I feel that if ten daily intramuscular doses of 50 mg. each fail to bring about some improvement, further testosterone therapy is useless and substitution of other forms of physical treatment is to be recommended. Testosterone is particularly useful in aborting minor relapses after otherwise successful electroshock treatments.

Testosterone may be combined with estrogen therapy in women instead of combining progesterone with estrogen. Medication useful in forestalling minor premenstrual depressions is, for instance, premarin 0.625 mg. combined with methyltestosterone 5 mg., twice daily by mouth, for twelve days prior to the period and for the first two days of the period. A similar, somewhat stronger helpful combination is to give estrogen in the form of eticylol or estinyl, 0.02 mg. daily for eighteen days after the period or for fourteen days prior to the period, and to give progesterone in the form of pranone, 10 mg. daily orally for six days prior to the period. I have also used pranone alone, 10 mg. every other day or twice weekly by mouth, for the treatment of anxious depressions that had remained partly refractory to combined convulsive-nonconvulsive electroshock treatments. The precise dose should be adapted to the individual case, preferably after consultation with an endocrinologist.

Testosterone therapy, 10 mg. three times daily in the form of methandrenolinguets, is also a useful adjunct in the therapy of frigidity in women. Another useful adjunct in the treatment of dysmenorrhea not related to endocrine disturbances is parpanit, 12.5 mg. four times a day orally, to be administered during the premenstrual period and throughout the period of menstruation.

Corticotropin (ACTH) and cortisone may produce euphoria and animadisturbance.¹¹⁵ Their use is therefore contraindicated in mental disease (see Chapter XVIII).

Thyroid Medication. The role of the thyroid in producing profound alterations in the mental state—cretinism in hypothyroidism, and states of

anxious agitation in hyperthyroidism (Graves' disease)—is well recognized. The latter particularly constitutes an important differential diagnostic problem, and the psychosomatic aspects of Graves' disease also deserve the psychiatrist's therapeutic attention. In view of this marked interrelationship between thyroid and nervous system, it is surprising that the thyroid is only rarely implicated in the vast majority of the so-called functional psychoses and neuroses that come to the psychiatrist for treatment. Even though the incidence of disturbances of the thyroid in these conditions is rare, the possibility of thyroid disease as a causative or contributing factor should always be kept in mind and appropriate diagnostic measures should be undertaken. Myxedema psychoses with confusional or paranoid symptomatology resembling presenile states not only clinically but also in some of their pathologic aspects have been described by Schnitzler.¹¹⁷

I once treated a woman, aged 52, for a severe schizophreniform psychosis with paranoid and catatonic features. Shortly after the onset of her psychosis she had elsewhere been given twenty electroshock treatments, between June 27 and September 5, 1947, with only brief transitory therapeutic benefit. When I saw her in a completely relapsed state on October 15, 1947, I was impressed by the fact that the skin of her face and arms was rather dry and that the density of her subcutaneous tissue was markedly increased. She showed the scar of a subtotal thyroidectomy that had been carried out fifteen years previously. Although her basal metabolic rate was within normal limits and her blood cholesterol was only moderately increased—230 mg. per cent—I felt the diagnosis of mild chronic post-thyroidectomy myxedema was warranted on clinical grounds. I placed her on a small amount of thyroid medication (1 grain daily), in addition to giving her a second intensive course of electroshock treatments. Eighteen treatments, administered between October 16 and December 2, 1947, together with continued thyroid medication, brought about a state of social recovery which gained further momentum after discharge and graduated into a state of complete recovery by February, 1948. Thyroid medication was continued after her discharge, and she has maintained her state of complete recovery consistently since that time. Her husband stated that he realized after her recovery that his wife must have been sick for at least ten years, for long before the apparent onset of her recent illness she had suffered from frequent and prolonged depressive and irritable mood swings, which had not recurred since recovery. I felt that while thyroid deficiency could not be regarded as the direct single cause of a schizophreniform psychosis of this kind, there was, nevertheless, suggestive evidence that it may have been a predisposing factor in this case as well as in other similar ones.

Although hypothyroidism is not a regular feature in the clinical picture of depression, the two states have certain signs in common, such as dry skin and elevated blood cholesterol. There is, however, no evidence that thyroid deficiency plays an etiologic role in depressions in general. Nevertheless, in some mild midlife depressions, especially in those occurring in a setting of fatigue and strain, a prescription of 1 grain of thyroid extract per day may be of decisive clinical benefit. In certain chronic neuroses manifested by depression, anxiety, and fatigue, however, one does not infrequently encounter cases of subclinical myxedema which can be greatly helped by thyroid medication.

An illustrative case is that of a young woman, aged 28, whom I had been treating by psychotherapy for three years with negligible results, and who

prior to that had been treated by psychoanalytic therapy elsewhere without having made any improvement. It gradually impressed me (somewhat late, I must admit) that her eyelids were unusually puffy, and on questioning the patient I learned that she had noticed episodes of occasional puffiness of the dorsa of her feet which she had dismissed, attributing it to her shoes. The skin of her hands and forearms was unusually dry and coarse, in marked contrast to the skin texture of her face and neck. Her pulse rate was 56 and her blood pressure 98/80. I then referred her to an endocrinologist who found that her basal metabolic rate was -38 per cent, and her blood cholesterol, 476 mg. per cent. The endocrinologist also found hypoferric anemia (hemoglobin 10.5 gm.). His findings warranted the diagnosis of severe hypothyroidism and hypoferrism consistent with hypothyroidism. Thyroid as well as iron was prescribed. Within a month, her basal metabolism rose to -5 per cent. Her mental symptoms showed marked improvement, but two months later she relapsed into her old neurotic anxious depressed state in spite of continued medication. Psychotherapy was therefore continued and for a time reinforced by nonconvulsive electric stimulation treatments. After a year of psychotherapy and thyroid medication she was greatly improved; her basal metabolism had risen to plus 1 per cent. Six months later she achieved full clinical recovery which has remained sustained since that time.

The use of extracts of the *pineal gland* for the treatment of mental diseases was first introduced by Pilcz,¹¹³ Lampl,¹⁰⁹ and Parhon and Tomorug.¹¹² Recently, Altschule and his coworkers¹⁰⁷ reevaluated its use in manic-depressive as well as in schizophrenic psychoses and found encouraging clinical and laboratory evidence of therapeutic action; there was clinical improvement as well as a rise in blood glutathione and eosinophils similar to the rise after electroshock treatment.^{105, 106}

ELECTRIC TREATMENT

Electric treatment may be administered through electrodes placed in bifrontal (Fig. 64A) or bitemporoparietal (Fig. 64C) position. In the

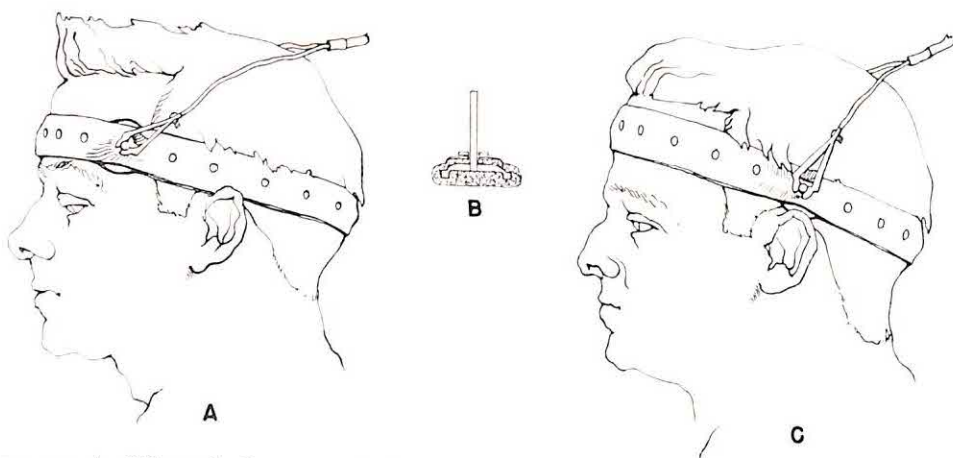


Fig. 64. A. Bifrontal placement of electrodes. B. Cross section of Nylon sponge-covered electrode. C. Bitemporoparietal placement of electrodes.

latter position each electrode should be centered 1 inch above the upper attachment of the scapha of the external ear. Convulsive treatment is usually applied through bifrontal and nonconvulsive treatment through

bitemporoparietal leads which are held in place by an adjustable rubber strap. Combined convulsive-nonconvulsive treatment may be given through either bifrontal or bitemporoparietal leads. It is my custom to give the briefer variant of combined treatment (2 minute treatment) through bifrontal leads while I give the longer variants of treatment (3 to 6 minute treatments) through bitemporoparietal leads. For still longer combined treatments (electric coma) I use a set of movable electrodes (Fig. 65) attached to handles which an assistant can move during treat-

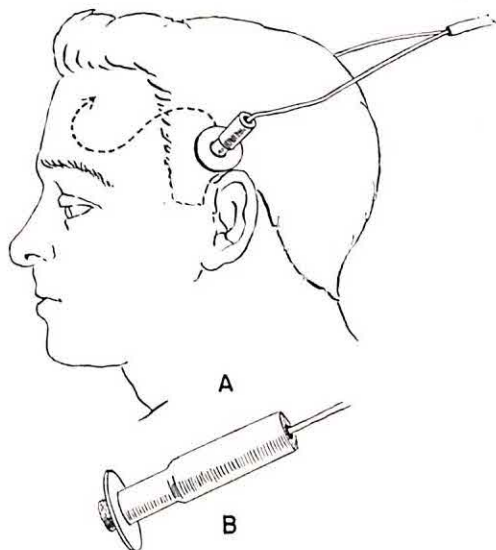


Fig. 65. Manually movable electrodes for use in electric coma therapy. A. The recommended excursions of the movable electrodes, indicated by a broken line. B. Lateral view of an electrode.

ment. Moving these electrodes from a bitemporoparietal position to a biparietal position and forward into a lower frontal position and then to an upper frontal position—following an S-pattern—lessens the intensity of the stimulation in a slight gradual manner, thus allowing modifications of current intensity in addition to those provided by the control dials of the machine.

The electrodes for the standard alternating current machines providing convulsive treatment currents consist of simple metal disks $2\frac{1}{2}$ inches in diameter. In applying these electrodes it is essential to use sufficient electrode jelly in order to provide good contact. For unidirectional current machines which require longer individual periods of treatment I use electrodes covered with nylon sponge (Fig. 64B) $1\frac{1}{2}$ inches in diameter and $\frac{3}{8}$ inch in thickness. These electrodes must be soaked in physiologic saline solution or in a weak solution of bicarbonate of soda (a knife point in a cup of water) prior to application. In addition I prefer to treat the skin at the prospective site of application of current with ample amounts of electrode jelly which should be rubbed gently into the skin in order to break down the outer oily film of the skin which is the greatest obstacle to adequate contact. In addition some excess of electrode jelly should be applied over the areas where treatment is to be given. This pretreatment

of the skin is important in order to insure that skin resistance is sufficiently low to avoid burns at the points of contact.

False teeth and any other foreign bodies should of course be removed from the mouth before treatment; gum and remnants of candy should always be inquired about or looked for.

The patient to be treated by convulsive treatment should be placed on a comfortable but firm couch or bed. Sandbags or fracture boards are generally unnecessary. If the treatment is to be given on a hospital bed that may tend to sag, however, a fracture board should be placed between the springs and the mattress. The thoracic spine should be in lordosis (Friedman, Brett, and Vogt¹²⁵). This can be brought about by placing a pillow under the small of the back. A second pillow should be placed under the head and shoulder and a third pillow beneath the knees (Figs. 29–31, pp. 115–116). The patient should be relaxed before treatment by appropriate conversation and supportive attitude. I have used the preambles of hypnotic suggestion with good success. More will be said about this in

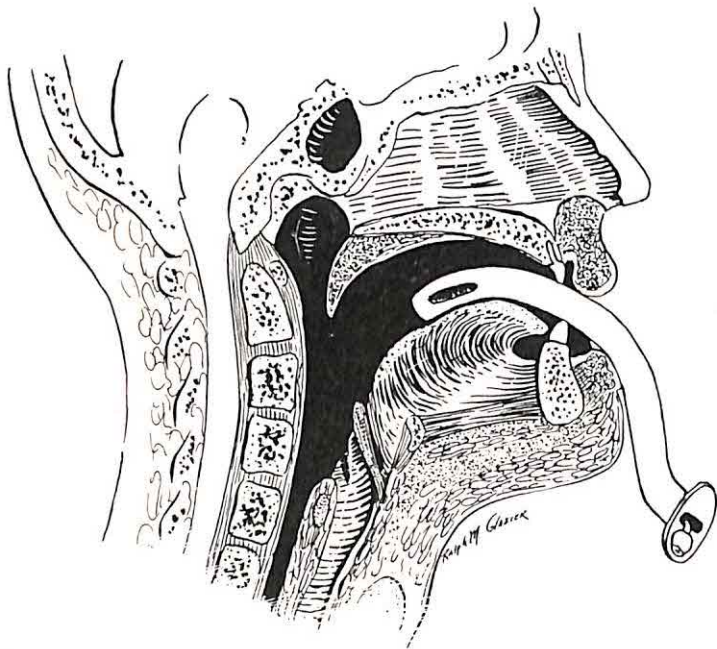


Fig. 66. A Guedel airway used as a tongue protector during convulsive electric treatment.

the chapter on preparation of the patient for treatment (Chapter XV).

The assisting nurse should place her hands on the shoulders of the patient, supporting partly the shoulder, partly the head of the humerus, fixing these parts firmly on the bed. When fixed electrodes are used, the doctor holds the chin of the patient with his right hand, at the same time holding the mouth gag in place, while he operates the machine with the left hand; or he may delegate the holding of the chin and mouth gag to a second nurse. When movable head electrodes are used, these should be entrusted to a nurse or assistant, while the doctor operates the machine and the mouth gag. As to choice of suitable mouth gags, I prefer to use a Guedel Rubber Airway as a mouth gag (Fig. 66) because it can be con-

veniently pushed back and used as an airway (Fig. 67) as soon as the patient becomes limp at the end of a seizure. The reason for this preference is the fact that the falling back of a large tongue may obstruct the air passage sufficiently to produce marked dyspnea and cyanosis, thus giving the false impression of severe laryngospasm. This can be prevented easily by immediately sliding in an airway as soon as the convulsion ceases. There is no more quick and convenient way to accomplish this than by using the back part of the airway as a mouth gag during the convulsion; the airway is then already between the teeth and above the tongue, requiring only one swift movement to slide it back as soon as the convulsion is over. I have repeatedly made the observation that patients who gave the impression of suffering from severe laryngospasm after each convulsive electroshock when the conventional felt pads were used as tongue protectors during the treatment, would start breathing immediately without obstruction when an airway was introduced promptly in the manner described above.

For nonconvulsive treatment one pillow under the head is sufficient; the nurse should slide the airway between the teeth and above the tongue as soon as the patient drops off to sleep, and hold chin and airway. The shoulders need not be restrained.

The only type of treatment during which it is advisable to have the

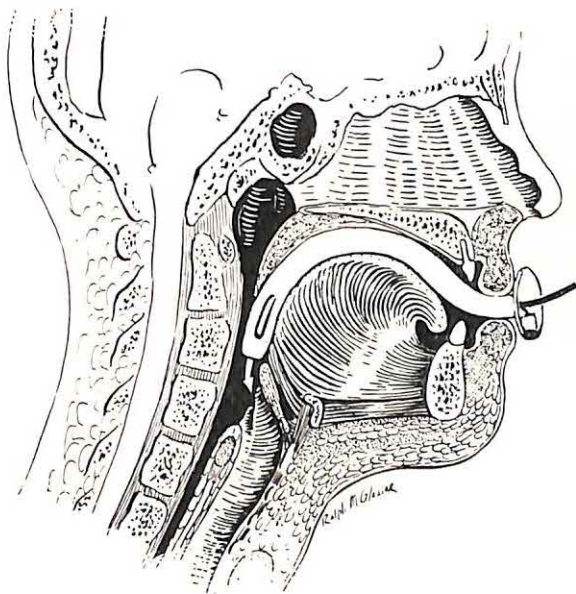


Fig. 67. The Guedel airway inserted immediately at termination of the seizure.

airway fully inserted throughout the nonconvulsive phase of treatment is the most prolonged type of combined treatment, namely electric coma.

The number of treatments which should be prescribed in an individual case cannot be foretold. The average number of treatments in uncomplicated cases is 7.5; atypical cases, especially depressions complicated by anxiety, schizophreniform and schizophrenic illnesses, frequently require a longer course of treatment, usually up to 20, sometimes up to 30 or

more, the average being 16.3. The largest number of treatments I have ever given in one continuous course was 39 treatments. This does not include cases on maintenance treatment (Stevenson and Geoghegan¹⁴³) given at weekly and wider spaced intervals, of which I gave up to 42 to any one patient. One of my patients, suffering from an involutional depression, was subject to frequent severe relapses; since the family rejected the recommendation of lobotomy there was no alternative but to give more treatments, which were necessary as lifesaving measures, because she refused all food during her depressive attacks and lost weight rapidly and alarmingly each time. The total number of treatments throughout all of her courses was 52, given in 7 series. The patient's final response was a state of apparent complete recovery which has held up well, longer than her previous healthy intervals which continued three to five months each.

A suggestive indication as to the number of treatments probably ultimately required for a good therapeutic result may sometimes become apparent during the course of treatment. I have found that the number of treatments required for the patient to make his first decisive step toward improvement not infrequently indicates the halfway mark in the total number required for full recovery. There are, of course exceptions to this suggestive rule, but by and large it has proven helpful in giving anxious relatives a realistic goal as a focus for their expectations.

The spacing of treatments should be close at first, and increasingly wide later. In general I give the first three treatments on a daily schedule, subsequent treatments until considerable improvement is made, on alternate days. From then on I increase the interval by a day each time, or after every other treatment, until the interval reaches a week, provided the patient holds his improvement well during the lengthening intervals; if not, I again shorten the intervals. I generally terminate the electric treatment series by the time it has become possible to let the patient go for a week without treatment, and without his showing distress or relapse. Variations of this schedule must be made, of course. If a patient improves considerably after the first or second treatment, wider spacing of treatments should be begun earlier. Wider spacing is also indicated when the physical condition of the patient may make it inadvisable to impose daily of maintenance treatments a week or longer apart may be added at the termination of the intensive course of treatment. In general, we must always adapt the schedule of treatment to the progress of the patient. I sometimes compare electric treatment to the manner in which one pushes a car stalled because of battery failure. The push should be sufficiently strong to make the motor turn over, but as soon as the car is running, the aiding car should hold back, following at a close distance to reapply the push if needed.

The important point is to treat the patient until the patient and his relatives agree that he has been restored to the status of "his own best self," and until his autonomic response pattern (Funkenstein test) has become normal. To stop at any time before clinical recovery and recovery of the reactivity of his autonomic nervous system are complete merely invites a long and troublesome period of post-treatment turmoil and probable eventual relapse, although a few fortunate patients do manage to struggle

through successfully to recovery even after an inadequate course of treatments.

There has been a tendency to more rigid pronouncements as to the best setting in which the treatment should be given—whether hospital in-patient, hospital out-patient, or office—than is warranted in view of the great variability of conditions, mental states, and personalities of patients to be treated. There is no doubt that for the agitated suicidal depressive the hospital is the best, in fact the only, place that can adequately fulfill the needs of the situation. On the other hand, the fact that convalescent patients should be allowed to complete their number of treatments on an out-patient basis has become increasingly and generally recognized (Gayle and Campbell¹²⁷). Out-patient or office treatment, however, can play an equally important role at the beginning of the treatment series, especially when we deal with a ruminative obsessive personality whose hostility would be unnecessarily aroused by removal from his customary setting and the blow to his pride which hospitalization frequently entails in these personalities. In cases with these and similar problems, arrangements other than hospitalization, namely out-patient treatment in an out-patient department or in the office, or even home treatment under certain circumstances, may be preferred. Sometimes a few treatments given under such circumstances may sufficiently soften the turmoil of the patient so that he is then willing to submit to hospitalization without excessive feelings of degradation, despair, and failure. Borderline depressions, especially of those in which there is an admixture of anxiety, do best in the setting of office treatment where there is more opportunity for individual psychotherapy and resulting good rapport, undisturbed by the disturbing aspects of a busy out-patient department. Needless to say, an office treatment room should be as well appointed with safety equipment as the treatment room of any first-rate hospital. Office treatment also requires the attendance of a graduate nurse. More about this will be said in the chapter on the role of the nurse during physical treatment (p. 418).

Nonconvulsive (Akinetic) Treatment

This treatment is given with the Reiter CW 47 machine through bi-temporoparietal leads. The treatment lasts from 7 to 12 minutes, and is initiated under pentothal anesthesia. I prefer to inject a 5 per cent solution of pentothal sodium at a fairly rapid rate, usually administering 4 to 6 grains over a period of 23 to 30 seconds, until the patient drops off to sleep. As soon as the patient ceases counting, the injection is stopped for 5 to 30 seconds, while the needle is still held in place, and the depth of anesthesia is tested by turning the current on at the average level of 2 milliamperes (Figs. 68 and 69). If the patient winces or withdraws from this small current more pentothal is injected until the patient tolerates 2 milliamperes (with spikes up to 80 milliamperes) without any motor response. The current is then promptly raised (within 2 to 4 seconds) to an average current of 10 milliamperes (with spikes up to 250 milliamperes) (Figs. 68 and 70). At this level respiration will become suppressed, at first completely, soon less and less completely. Kymograph recordings show that slight respiratory oscillations are coming through increasingly as current is maintained at that level (Fig. 68), usually within 20 to 40 seconds after the start of the stimulation. The current is allowed to stay at

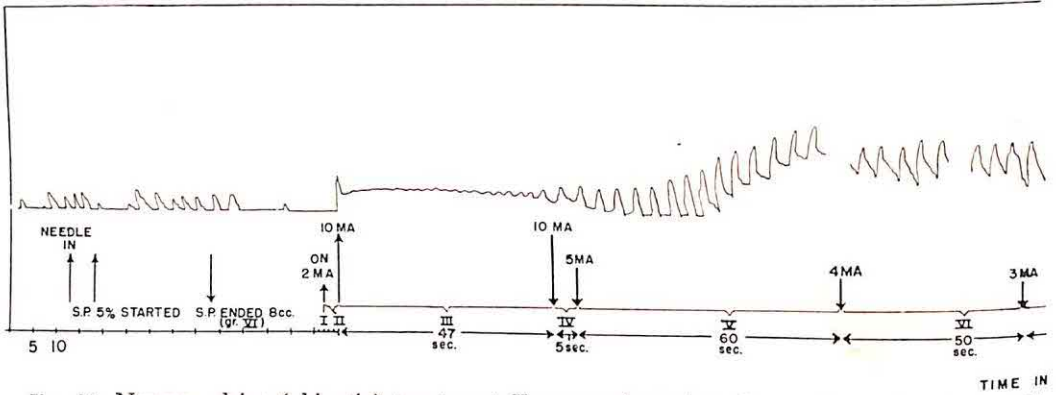


Fig. 68. Nonconvulsive (akinetic) treatment. Kymograph tracing obtained from the chest wall three arrows indicate preparation, start, and termination of the pentothal sodium injection. The re-raising, arrows pointing downward indicate lowering of current. The Roman numerals corre- 69-81. The duration of each of these phases is indicated below the Roman numerals, and on the showing no marked change in the respirograph pattern have been omitted, these omissions being indicated at the lower margin of the figure.

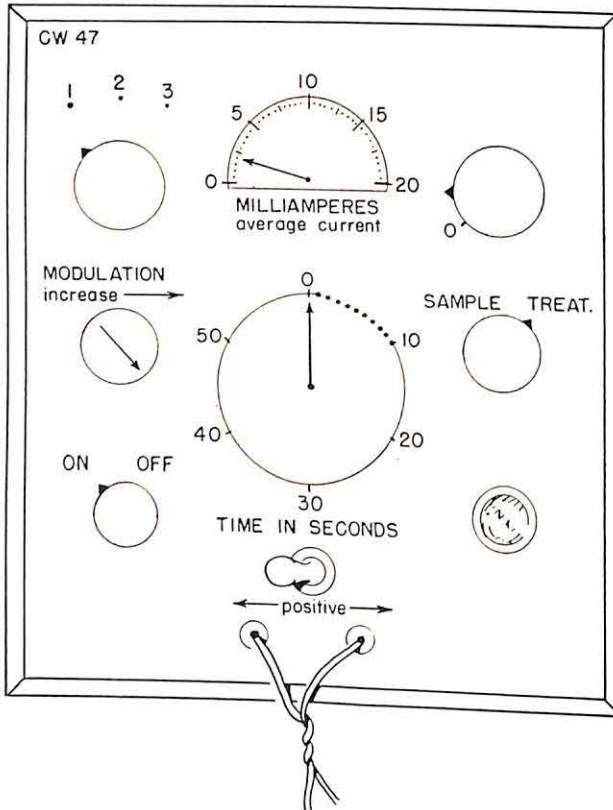
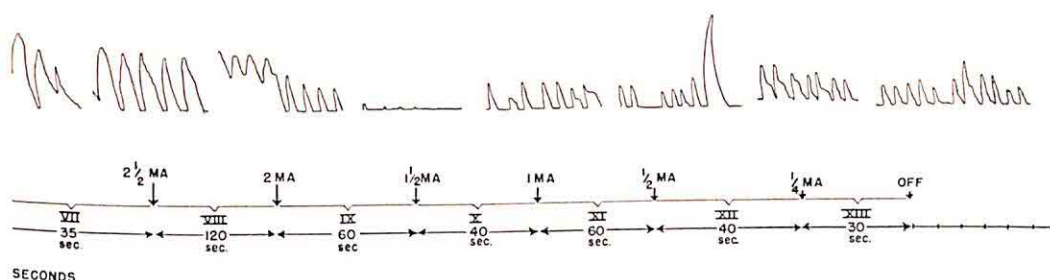


Fig. 69. Nonconvulsive (akinetic) treatment. This and the following 12 dial-face diagrams illustrate the treatment administered with the Reiter CW 47 machine. Step I. Set current control dial at average current of 2 MA.

this level of 10 milliamperes of average current for about 47 seconds (Figs. 68 and 71). It is then lowered sufficiently to allow respirations to come through fully, which will usually be at an average current of 5



SECONDS

(respirogram) of a patient before, during, and after non-convulsive electric treatment. The first maining arrows indicate start and alterations of flow of current. Arrows pointing upward indicate spond to the thirteen distinct steps of the procedure illustrated in the dial-face diagrams, Figs. time clock of the following dial-face diagrams. In view of the length of the treatment, sections indicated by gaps in the tracing. However, the full duration of each step of the procedure is

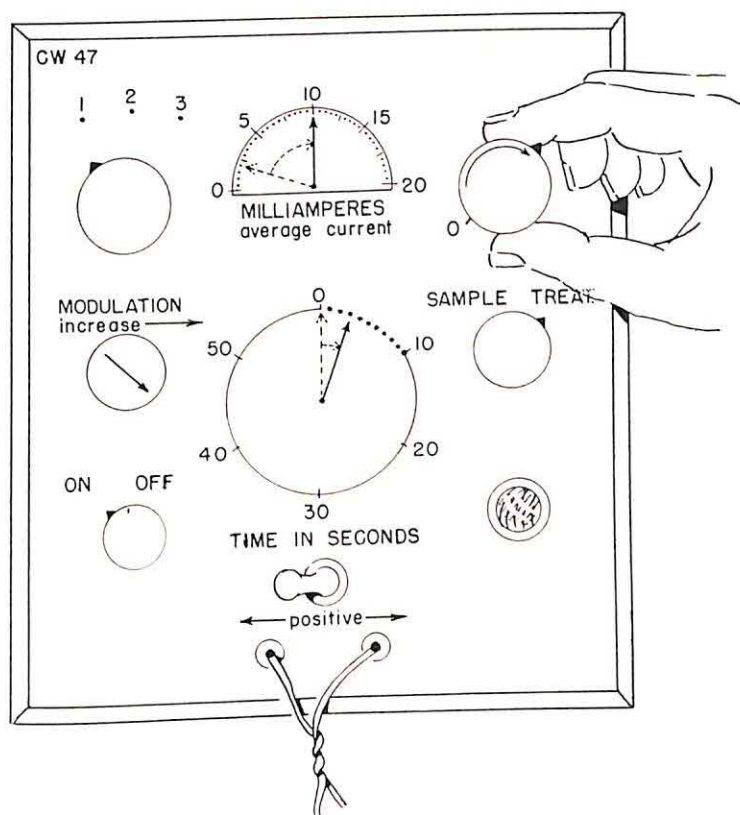


Fig. 70. Nonconvulsive (akinetic) treatment, Step II. Raise current within 3 seconds to 10 MA.

milliamperes (with spikes up to 160 milliamperes) (Figs. 68 and 72). Even if apnea has prevailed throughout the full first 50 seconds of the treatment, respirations promptly break through when the current is reduced to half, i.e., at 5 milliamperes of average current.

Stimulation is continued at 5 milliamperes for about one minute, during which time polarity should be reversed every 20 to 30 seconds (Fig. 73). However, the current should be lowered earlier—to average currents of 4, 3, or $2\frac{1}{2}$ milliamperes, dependent on the need indicated by the quality of respiration, i.e., if respiration should become labored at any of these levels. If all has gone well, the current should be reduced to 4 milliamperes after one minute and kept there as long as the patient breathes comfortably,

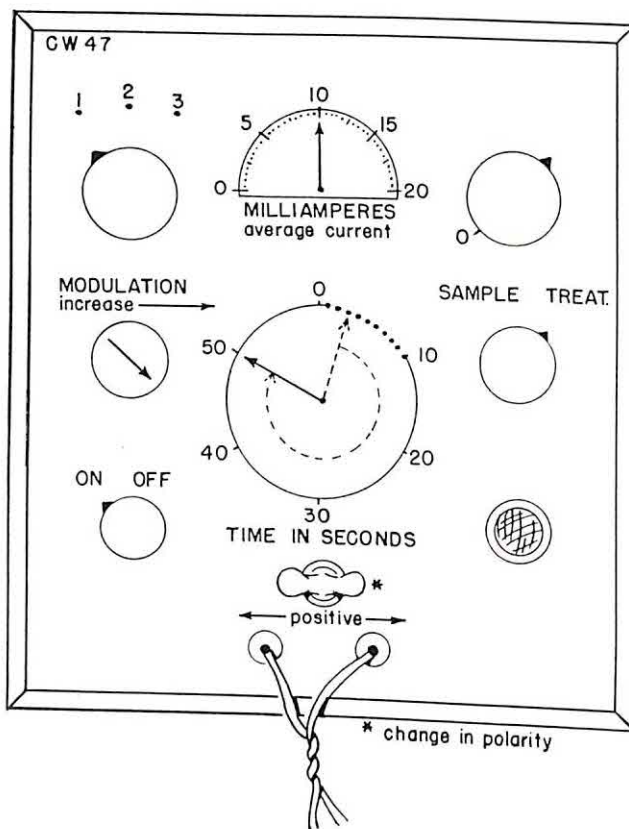


Fig. 71. Nonconvulsive (akinetic) treatment, Step III. Hold current at 10 MA for 47 seconds.

or until the patient awakens (Fig. 74). Generally patients will waken at the end of the third minute of treatment, the facial expression denoting that the current has become uncomfortable. The current should be gradually lowered to the precise point at which the current becomes comfortable to the patient (Figs. 75–81). The patient should be told to raise his right hand whenever the current becomes uncomfortable and the current must be lowered whenever he does so. This assures a comfortable state throughout the treatment. Sometimes the reduction of current by merely $\frac{1}{2}$ milliamperes will render the patient comfortable. However, 30 to 60 seconds later the patient will again complain, and the same procedure of lowering the current by $\frac{1}{2}$ milliamperes per step should be followed. This method should be continued until $\frac{1}{4}$ milliamperes is reached, at which time the current should be shut off. From average currents of about 2 milliamperes

on down patients are generally able to communicate with the doctor, and some abreaction may take place at this time, to become intensified at the end of the treatment. This, however, is not invariably the case, and is not necessary for therapeutic success. If pentothal anesthesia was excessively deep patients will wake slowly. In those cases more frequent changes in polarity or raising the current up again to 10 milliamperes for a few seconds will hasten the waking process.

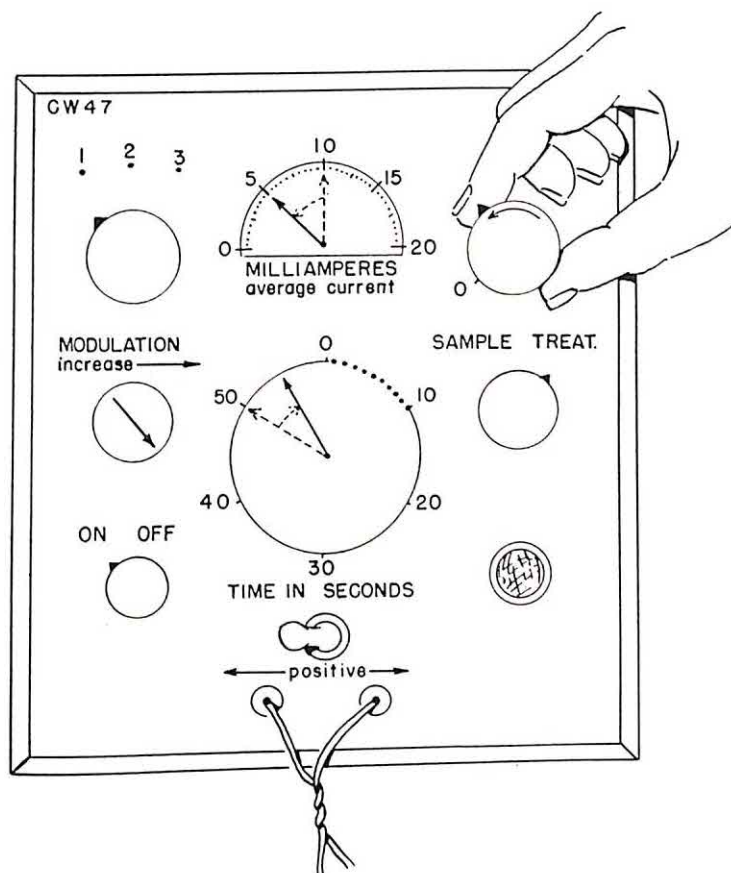


Fig. 72. Nonconvulsive (akinetic) treatment, Step IV. Reduce current to 5 MA within 5 seconds.

Directions for Nonconvulsive Electric Treatment. As soon as induction of anesthesia by pentothal sodium has been completed, the patient is ready for the electric treatment; however, the injection needle should be kept in place until tolerance of the first step of treatment has confirmed the adequacy of the anesthesia. The following are the precise steps and their duration recommended for this treatment:

Step I. Select No. 1 current and fast modulation, turning the modulation knob to the right as far as it will go. Keep No. 1 current and fast modulation pattern throughout treatment. Set the machine at an average current of 2 milliamperes. Turn on machine (Figs. 68 and 69). This step is the test for the adequacy of the depth of the anesthesia; if the patient winces or shows other signs of pain the current must be turned off im-

mediately and more pentothal must be given. If the patient shows no signs of pain, the needle should be withdrawn, and the patient is ready for the next step of treatment.

Step II. Raise the current swiftly (within 3 seconds) to 10 milliamperes (Figs. 68 and 70).

Step III. Keep current at an average level of 10 milliamperes for 47 seconds. During this time change polarity about every 20 seconds (Figs. 68 and

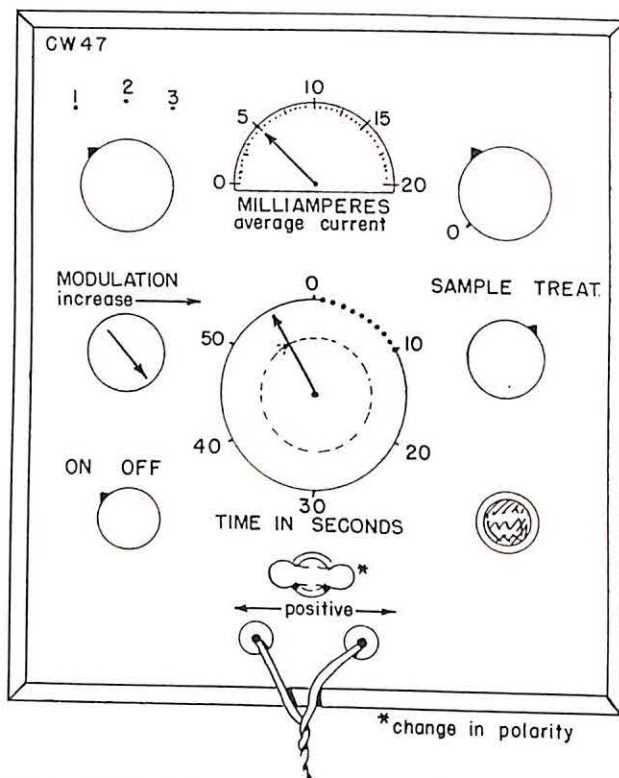


Fig. 73. Nonconvulsive (akinetic) treatment, Step V. Hold current at 5 MA for one full minute.

71). Respiration is usually suppressed at this level, but after about 20 to 40 seconds shallow respiratory oscillations will usually break through in spite of the maintenance of the current at the same level (Fig. 68).

Step IV. Reduce current from 10 milliamperes to 5 milliamperes within 5 seconds, provided the patient was breathing deeply either before this step was undertaken or when the average of 5 milliamperes was reached (Fig. 72). If the patient does not breathe deeply at 5 milliamperes, reduce the current still further to 4, 3 or $2\frac{1}{2}$ milliamperes, whatever is necessary to reestablish adequate respiration. If respiration remains inadequate, turn the modulation knob to slow, i.e., counterclockwise. There is usually no difficulty in reestablishing adequate respiration in nonconvulsive treatment at levels between $2\frac{1}{2}$ and 5 milliamperes of average current. Therefore, additional stimulating maneuvers which may be useful in combined treatments or in electric coma, such as raising the current swiftly to 10

milliamperes and lowering it to $2\frac{1}{2}$ milliamperes in quick succession, are usually not necessary in nonconvulsive treatment.

Step V. If respiration is adequate at 5 milliamperes keep current at that level for a full 60 seconds (Fig. 73). If the respiration remains quite free and adequate, the current may be kept at that level even longer. I have used periods of stimulation at that level varying from as short as 38 seconds to as long as 89 seconds. After about 30 to 40 seconds at this level, expira-

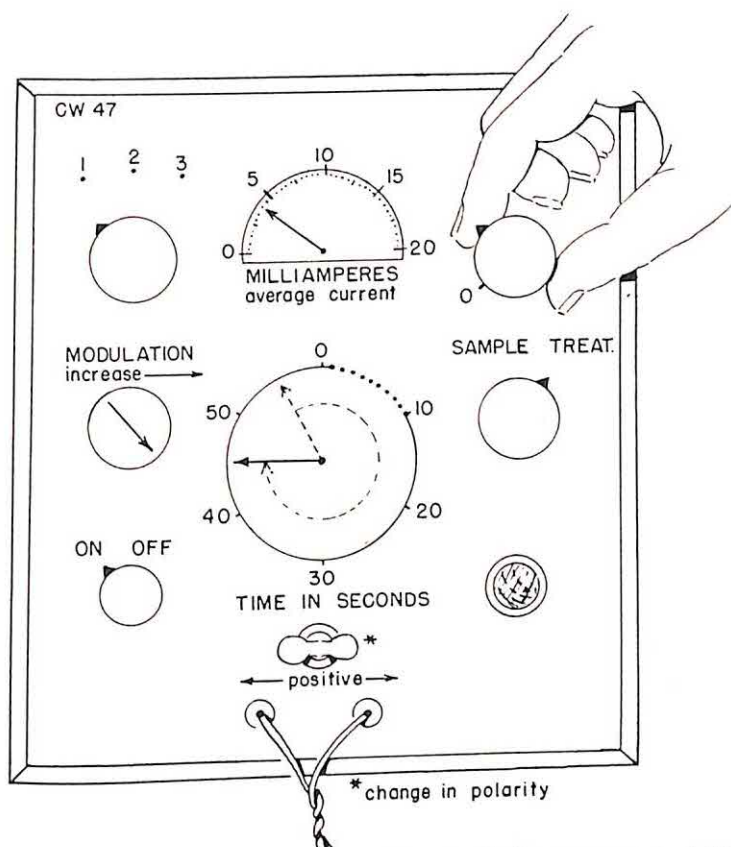


Fig. 74. Nonconvulsive (akinetetic) treatment, Step VI. Reduce current to 4 MA and hold there for 50 seconds.

tion may become inadequate (reduced in depth), associated with rise in intrathoracic pressure, due to laryngeal stridor (Fig. 68). If this occurs the current level must be lowered until stridor is relieved and expiration becomes free (Fig. 68).

Step VI. Lower current to 4 milliamperes and keep at this level for 50 seconds, provided respiration remains adequate and the patient feels no pain (Figs. 68 and 74).

Step VII. Lower current to 3 milliamperes and keep at that level for about 35 seconds (Fig. 75). This is about the time at which patients may begin to waken and to express pain. If that occurs, the current should instantly be lowered to a comfortable level. Lowering the current by as little as $\frac{1}{2}$ milliampere may make the patient comfortable.

Step VIII. Lower current to $2\frac{1}{2}$ milliamperes and keep at that level for about 2 full minutes (Fig. 76). If the patient has not yet awakened, he will tolerate stimulation at this level well. Under such circumstances I have kept the current at this level for as long as 3 minutes. If the patient indicates that the current is uncomfortable at this level, it should be instantly lowered. In such cases I have had to reduce stimulation at this level to as little as 25 seconds, but was able to get a full 3 minutes of stimulation at

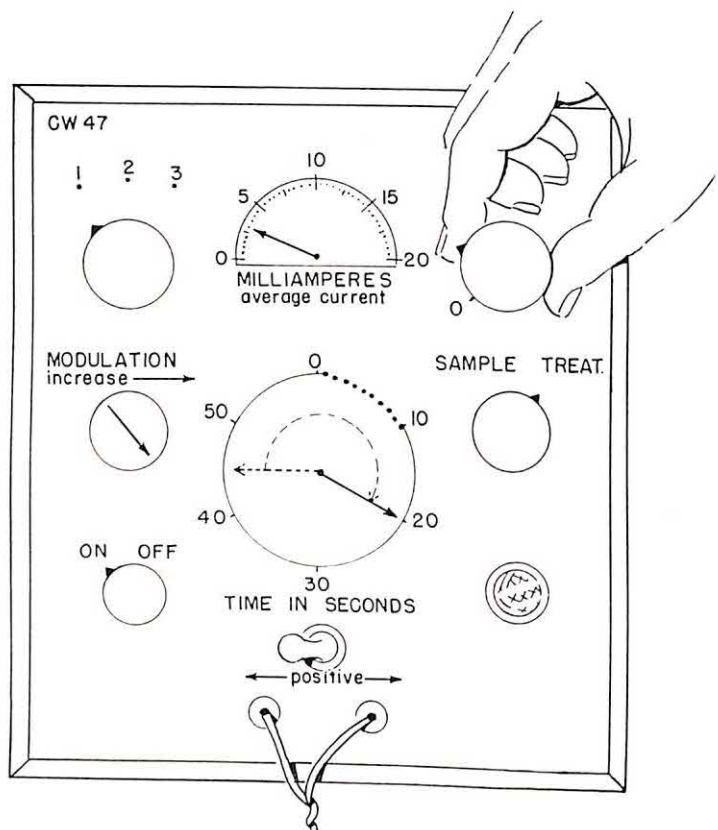


Fig. 75. Nonconvulsive (akinetic) treatment, Step VII. Reduce current to 3 MA and hold there for 35 seconds.

the next lower level, namely, 2 milliamperes. During this period polarity should be changed every 20 seconds. As soon as the patient awakens or feels pain, the amplitude of his expirations may become reduced. Lowering the current to the next lower level (2 milliamperes) instantly relieves this reflex-dyspnea (Fig. 68).

Step IX. Reduce the current to 2 milliamperes and keep at that level for about one minute (Figs. 68 and 77). Whenever pain comes to the fore, of the current, which will relieve the pain and free the respiratory activity.

At this level of nonconvulsive stimulation the patient will be able to let go of the mouth gag and to talk. Some abreaction may begin at that time. It is important to lower the current instantly when the patient gives the signal indicating that the current is becoming uncomfortable to him.

Step X. Reduce the current to $1\frac{1}{2}$ milliamperes and hold at that level for about 40 seconds (Figs. 68 and 78). Then, as soon as the patient indicates that the current has become uncomfortable, reduce the current to the next lower level.

Step XI. Reduce the current to 1 milliampere and keep at that level for 1 minute, unless the patient says or signals before the end of that time that the current has become uncomfortable (Fig. 79).

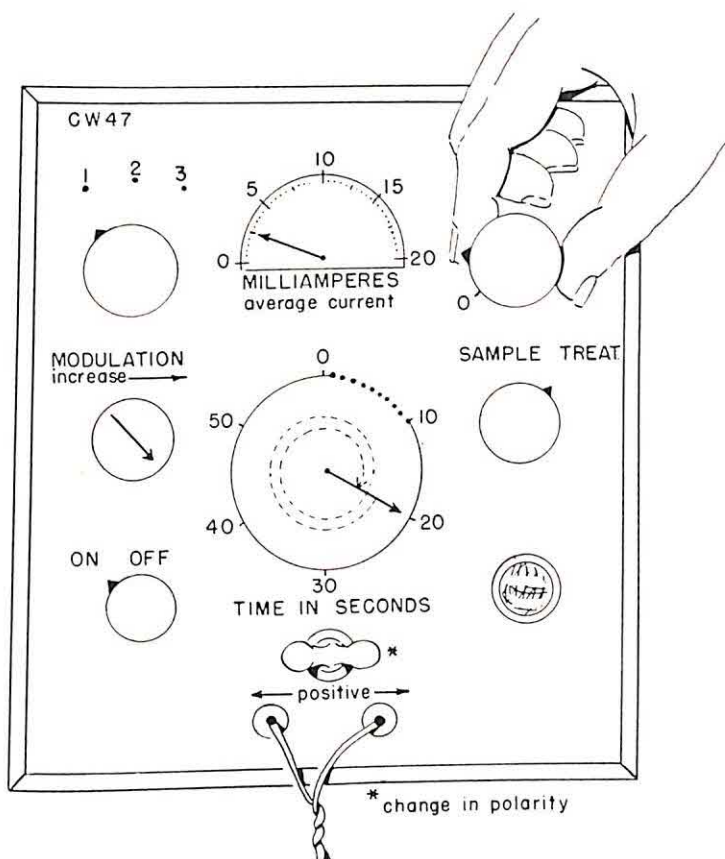


Fig. 76. Nonconvulsive (akinetic) treatment, Step VIII. Reduce current to $2\frac{1}{2}$ MA and hold there for 2 minutes.

Step XII. Reduce the current to $\frac{1}{2}$ milliampere and keep it at this level for about 40 seconds (Fig. 80), which is usually easily tolerated. There may be considerable abreaction during this time, sometimes punctuated by deep sighs (Fig. 68).

Step XIII. Reduce the current to $\frac{1}{4}$ milliampere and keep at that level for about 30 seconds at which time the current should be shut off (Figs. 68 and 81).

The total average time of this treatment as outlined above is approximately 9 minutes and 10 seconds. After completion of the treatment considerable time should be spent in psychotherapy, then the patient should be allowed to drop off to a refreshing sleep. Sometimes the patient will become sleepy after a fairly brief psychotherapeutic interview. In such cases

further therapy can be deferred until after the sleep. When treatment is given on alternate days, psychotherapeutic interviews should be held also on each day following treatment. The facilitation of psychotherapy by nonconvulsive treatment will be discussed in further detail in Chapter XV, page 405.

Combined Myoclonic-Nonconvulsive Treatment

The technique of myoclonic treatment is in all respects identical with that of nonconvulsive treatment, except that in Step II the current is raised from 2 milliamperes to 15 milliamperes (instead of only 10 milliamperes)

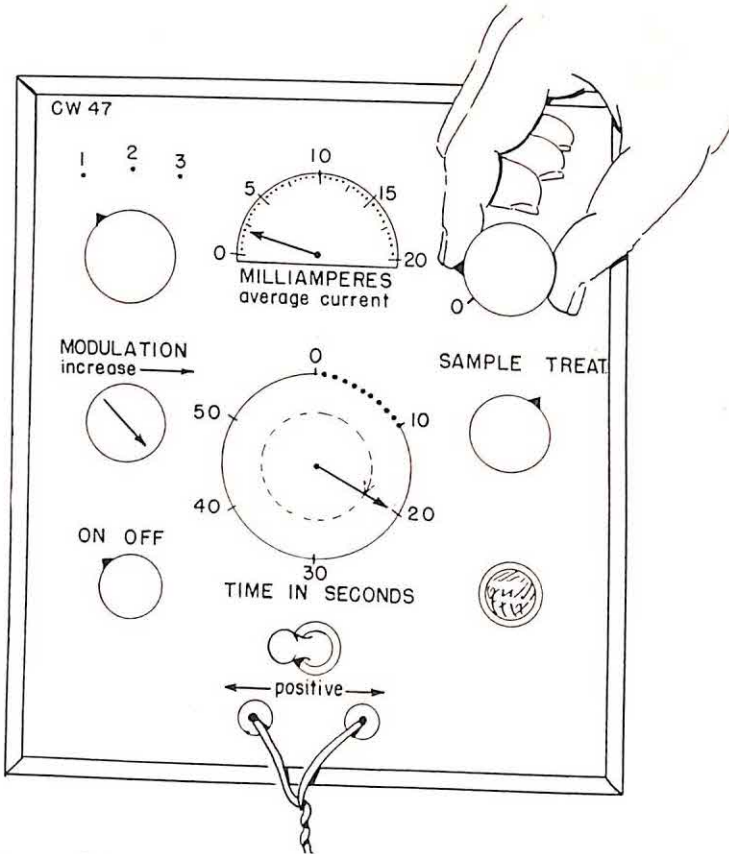


Fig. 77. Nonconvulsive (akinetic) treatment, Step IX. Reduce current to 2 MA and hold there for one minute.

and held at that level for 47 seconds (Figs. 82, 83, 84). Kymograph recordings show myoclonic twitching during that phase, which ceases as soon as the current is reduced (Fig. 82). Respirations may come through in spite of the superimposed myoclonic twitchings within 20 to 45 seconds after onset of the treatment (Fig. 16), although they may be delayed until the current is reduced (Fig. 82).

Step I. Test depth of anesthesia with an average current of 2 milliamperes, as in nonconvulsive treatment (Figs. 69 and 82).

Step II. Raise current within 3 seconds to an average current level of 15 milliamperes (with spikes up to 345 milliamperes) (Figs. 82 and 83).

Step III. Maintain average current at 15 milliamperes for 47 seconds. Myoclonus manifests itself at this level (Figs. 82 and 84).

Step IV. Reduce current from 15 milliamperes to 5 milliamperes within 10 seconds (Figs. 82 and 85).

Step V. Keep current at 5 milliamperes for 55 seconds. If breathing is not adequate at 5 milliamperes, reduce current immediately to 4 milliamperes or lower.

From here on the subsequent steps of treatment are entirely identical with Steps VI–XIII of nonconvulsive treatment (see preceding section).

Convulsive Treatment

Purely convulsive treatment, whether with alternating or unidirectional current, is best given through frontal leads. For treatment with alternating

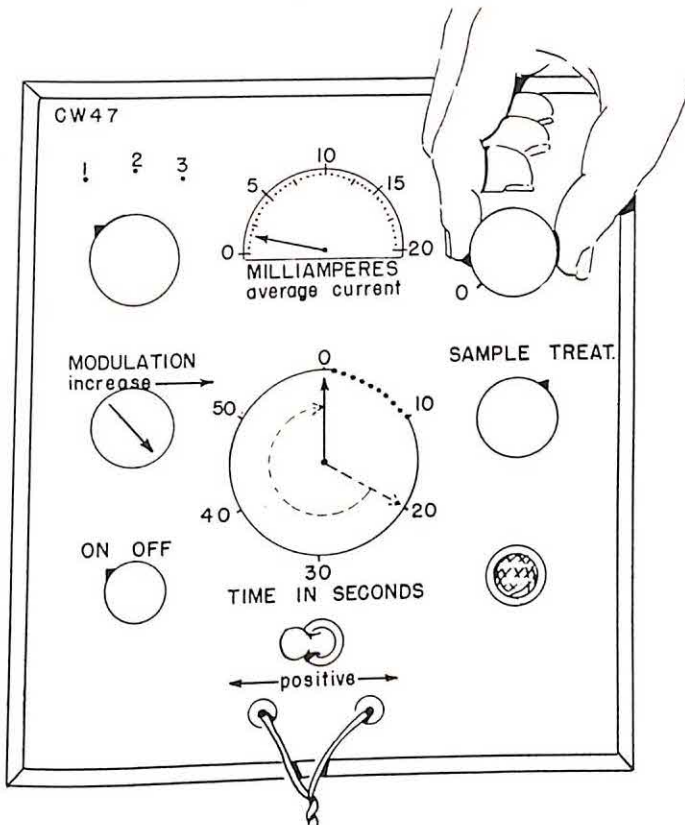


Fig. 78. Nonconvulsive (akinetic) treatment, Step X. Reduce current to 1½ MA and hold there for 40 seconds.

current a number of well built, safe, and reliable machines are available, such as the Medcraft machine, the Lektra machine, and numerous others. The Medcraft machine has the advantage of being particularly compact and easily portable. The dial face of the machine dispenses with an amperemeter, since the average amperage can be readily computed according to the formula: voltage divided by resistance equals amperage ($V/R=I$), provided that contacts are good. It is important for the sake of good contact to pretreat the skin by rubbing it with electrode jelly, in

addition to applying ample amounts of electrode jelly between skin and electrodes. A current of 120 volts applied with this machine (Fig. 86) will deliver average currents of 330 milliamperes, provided that the skin resistance including resistance of the machine and the leads is at a good average low of 360 ohms. Resistance of the human body decreases with increased perfection of contact, with increased duration of time of contact and of flow of current, and with increased voltage. It varies from a maximum of 50,000 to a minimum of 218 ohms. For the conditions prevailing

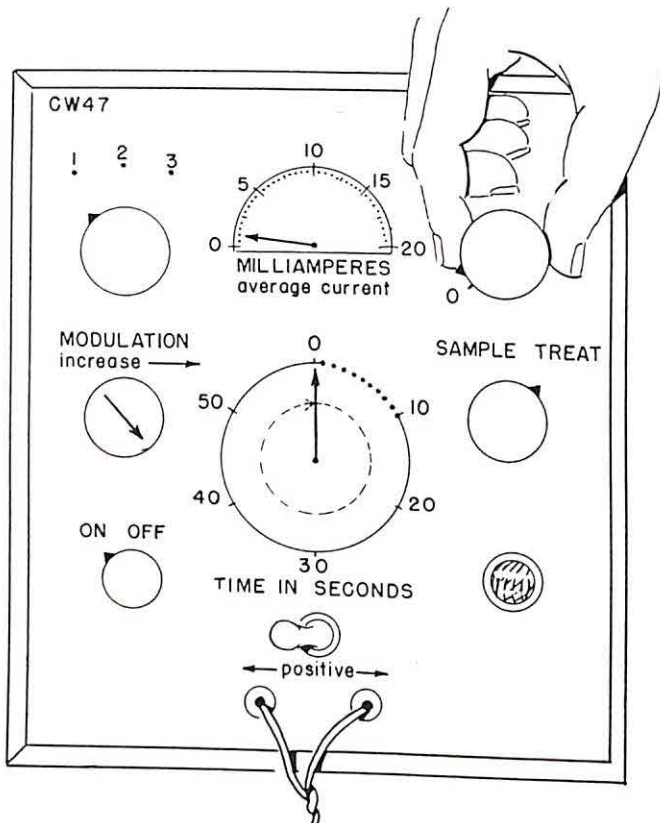


Fig. 79. Nonconvulsive (akinetic) treatment, Step XI. Reduce current to 1 MA and hold there for one minute.

with electric shock treatment, a resistance of 360 ohms is a suitably low one.

The Medcraft machine also is supplied with a glissando attachment, to be used according to the glissando technique developed by Tietz and her co-workers.¹⁴⁴ The glissando (slow rise) technique softens, or rather breaks tonic phase, by dividing it into two very brief bursts, interpolating an interval of relaxation (Fig. 87, compare with Fig. 17). Prior to treatment the push button will then release gradually increasing currents—which will build up within 0.6 of a second to the full strength of the current selected, e. g., 330 milliamperes, if the voltage selector was set at 120 volts.

This current will stay on for the amount of time selected on the time selector (labeled "Seconds"), which allows selections of times varying from 0.1 of a second to a full second. In general, stimulation times of 0.5 to 0.6 second (not including the glissando build-up period) will be adequate. If this current proves insufficient to produce a convulsion, the voltage selector as well as the time selector should be turned fully to the right, delivering a current of 400 milliamperes flowing for one second. With some alternating current machines, including also another Medcraft

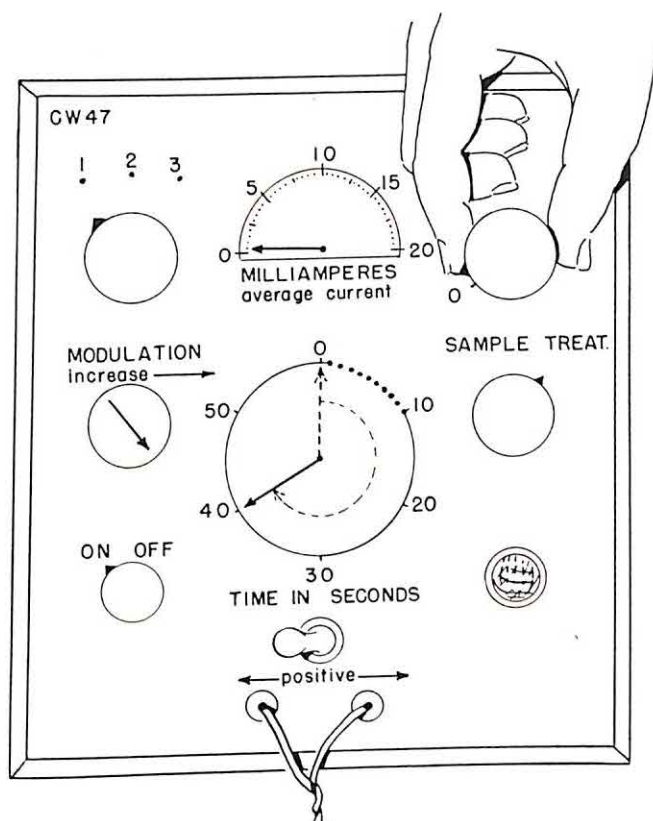


Fig. 80. Nonconvulsive (akinetic) treatment, Step XII. Reduce current to $\frac{1}{2}$ MA and hold there for 40 seconds.

model, currents up to 180 volts can be applied delivering average currents of 500 milliamperes.

On the Lektra machine (Fig. 88) the resistance of the patient can be measured by a test circuit (which is below the threshold that the patient can feel) and the desired amount of amperage can be applied. This helpful device, which was suggested by Myerson, Feldman, and Green,¹³⁷ and incorporated into the early Davis machine, has the advantage that imperfections of contact due to excessively oily skin, or application of insufficient amounts of electrode jelly, can be detected before treatment and subconvulsive shocks or shocks that the patient may feel or that may cause burns can thus be avoided. An experienced team, however, will rarely require this special safeguard, although it adds to the precision in applying desired

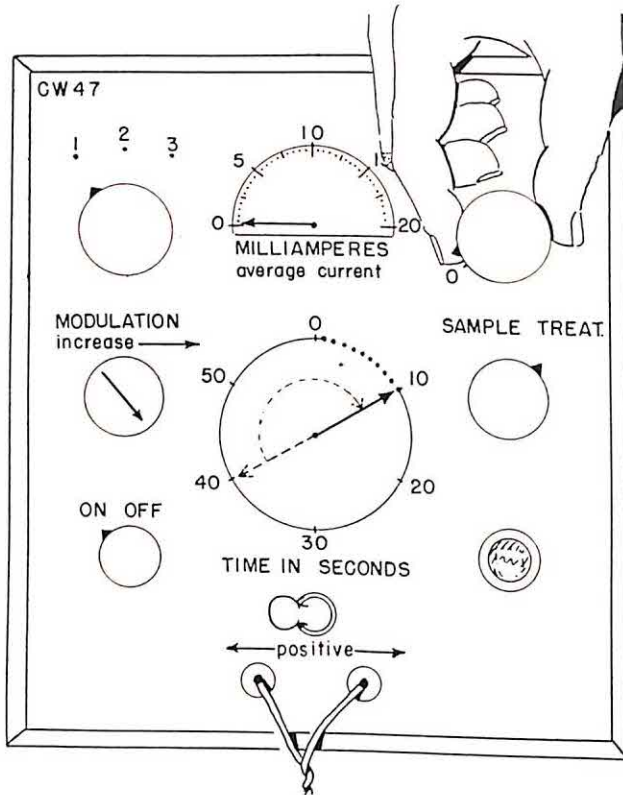


Fig. 81. Nonconvulsive (akinetic) treatment, Step XIII. Reduce current to $\frac{1}{4}$ MA, hold there for 30 seconds, then shut off current.

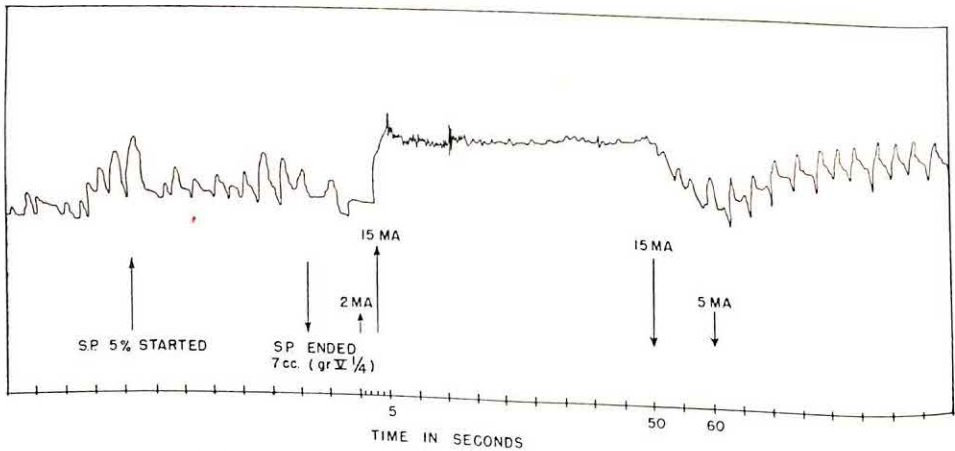


Fig. 82. The initial (myoclonic) phase of combined myoclonic-nonconvulsive electric treatment. Kymograph tracing (respirogram) obtained from the chest wall of a patient. The first two arrows denote the beginning and end of the pentothal sodium injection. The next two arrows pointing upward indicate the turning on and raising of the current, MA to 5 MA. The remainder of the treatment is identical to that shown in Fig. 68.

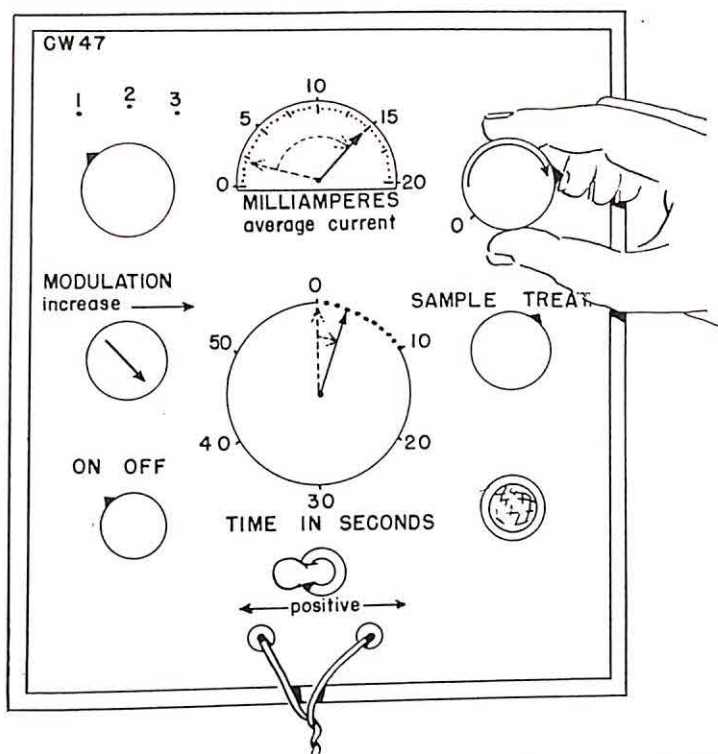


Fig. 83. Combined myoclonic-nonconvulsive treatment. This and the following 2 dial-face diagrams illustrate phases of treatment administered with the Reiter CW 47 machine. Step II. Raise current within 3 seconds from 2 MA to 15 MA.

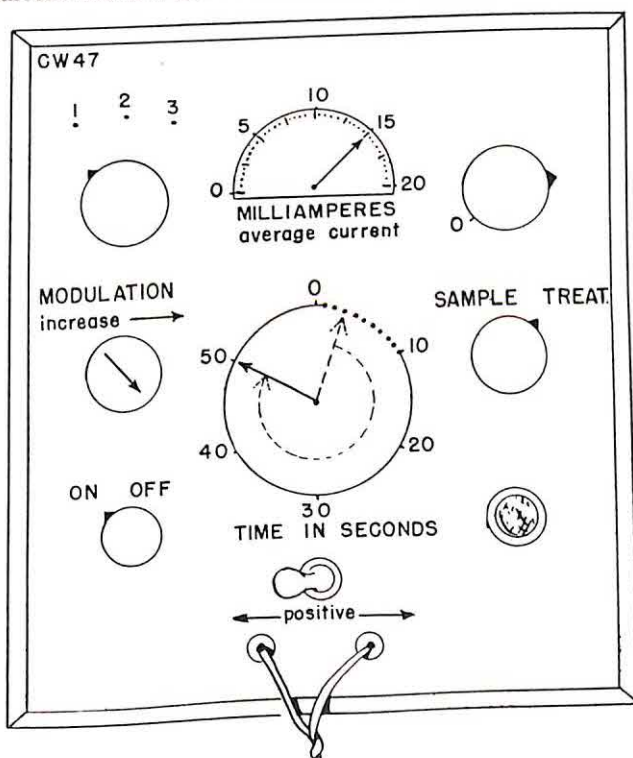


Fig. 84. Combined myoclonic-nonconvulsive treatment, Step III. Hold current at 15 MA for 47 seconds.

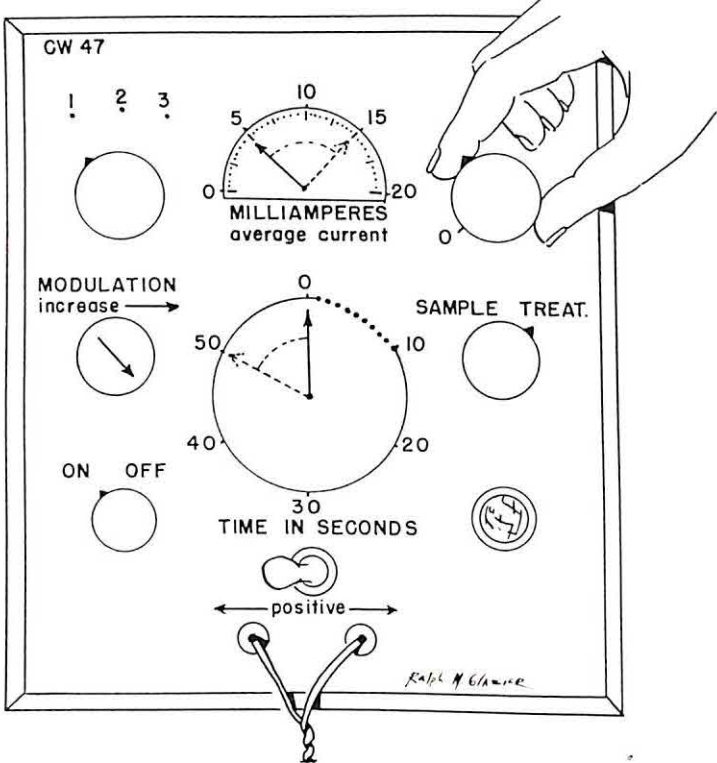


Fig. 85. Combined myoclonic-nonconvulsive treatment, Step IV. Reduce current to 5 MA within 10 seconds.

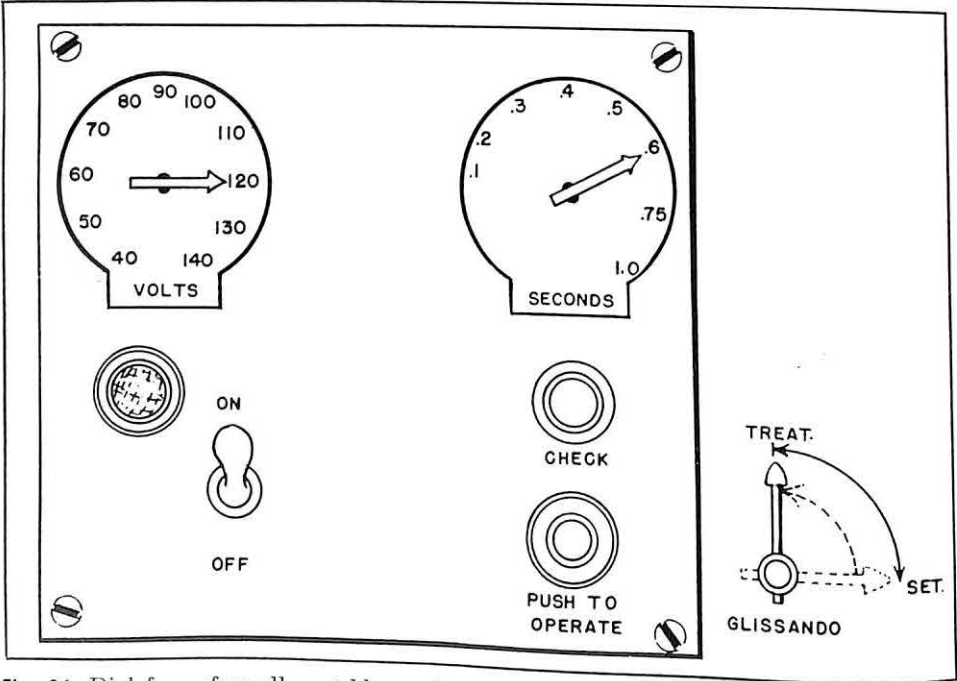


Fig. 86. Dial face of small portable machine for convulsive treatments with alternating current with "glissando" attachment (Medcraft).

intensity of stimulation. The Lektra machine also has a glissando attachment.

Convulsive treatment can also be administered by unidirectional currents either by means of the Liberson-Offner brief stimulus apparatus or by any of the Reiter type machines. Reiter's model CW 46 is well suited for convulsive treatment. Stimulation by currents varying from 5 milliamperes of average current (with spikes up to 370 milliamperes) to 20

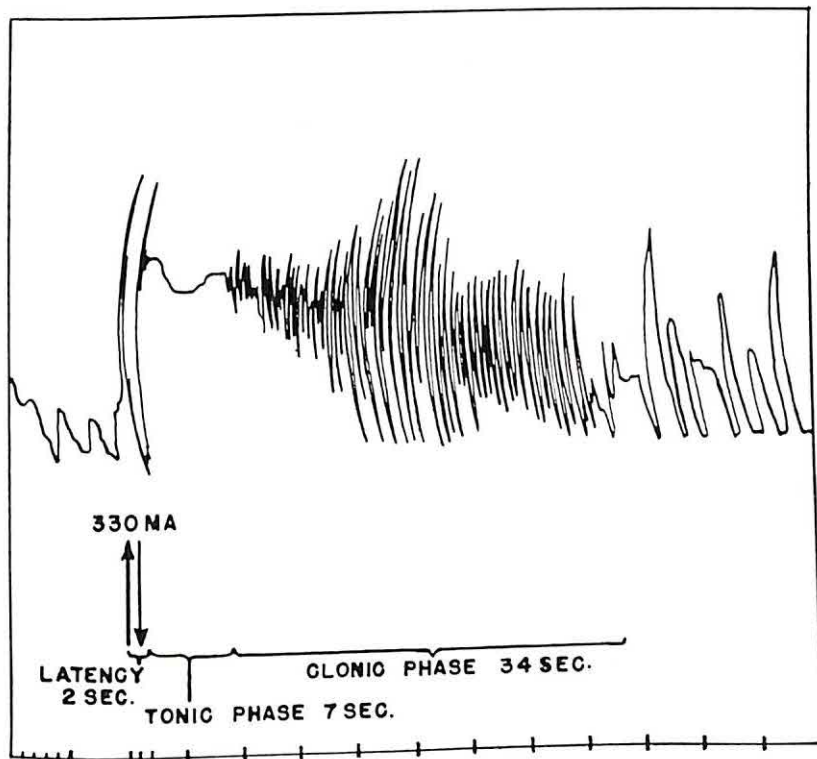


Fig. 87. Respirograph tracing of a patient undergoing convulsive electroshock treatment elicited by alternating current of 0.5 second duration, preceded by slow rise ("glissando") of 0.6 second duration. Medcraft machine. The first arrow, pointing upward, denotes the onset of the slow rise ("glissando"). The time at which the current reaches full strength (330 MA of average current) 0.6 second later is not separately indicated on the graph. The second arrow, pointing downward, indicates the time at which the current is shut off, 1.1 seconds after the beginning of the slow rise, and 0.5 second after the current had reached full strength.

milliamperes of average current (with spikes up to 1670 milliamperes) of 5 seconds' duration through bifrontal leads will elicit a tonic-clonic seizure in most patients. In some patients with high rheobase, stimulation may have to be continued up to 30 seconds to produce a tonic-clonic seizure. The greatest advantage of unidirectional currents is that the clonic phase can be entirely suppressed by maintaining the current at the convulsion-producing level throughout the time span of the convulsion, namely 54 seconds (in the manner described on p. 340).

One of the most important advantages of this technique is that it maintains peripheral vasodilatation as well, thus reducing the peripheral resistance that the heart must overcome during and after the termination of

the seizure. By gradually reducing the current from the 35th second of the stimulation onward, it is also possible by this method to reduce intra-thoracic pressure gradually, thus relieving the Valsalva phenomenon in easy stages (see Fig. 89). In this manner it is possible to reduce the inflow

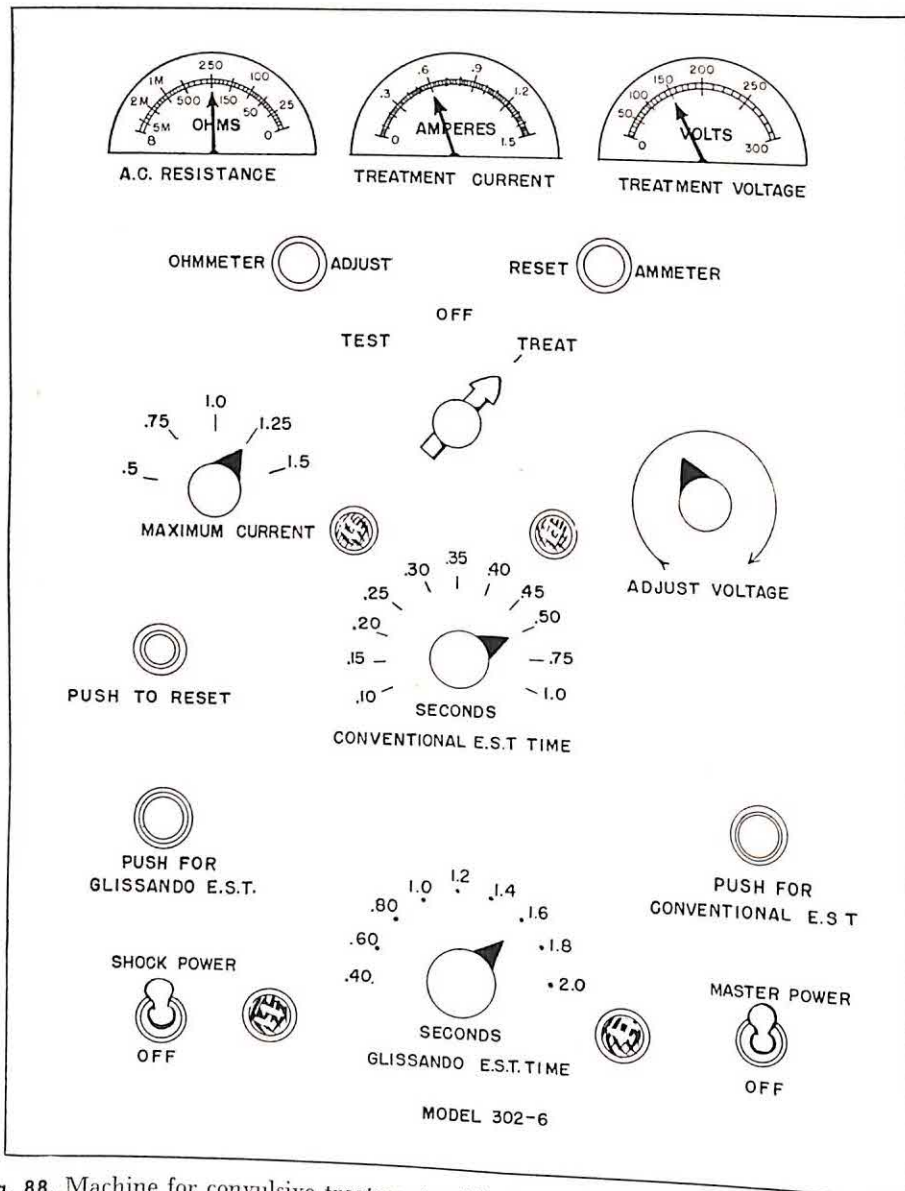


Fig. 88. Machine for convulsive treatments with alternating current, fitted with ampere-meter and test circuit for determining resistance (Lektra).

load of the heart during the postconvulsive period. This maneuver should always be resorted to in patients with myocardial impairment, coronary heart disease, and disturbances of the pacemaker mechanism of the heart. In addition, of course, to other measures which are recommended in Chapter X. I merely wish to reemphasize that postshock disturbance of heart

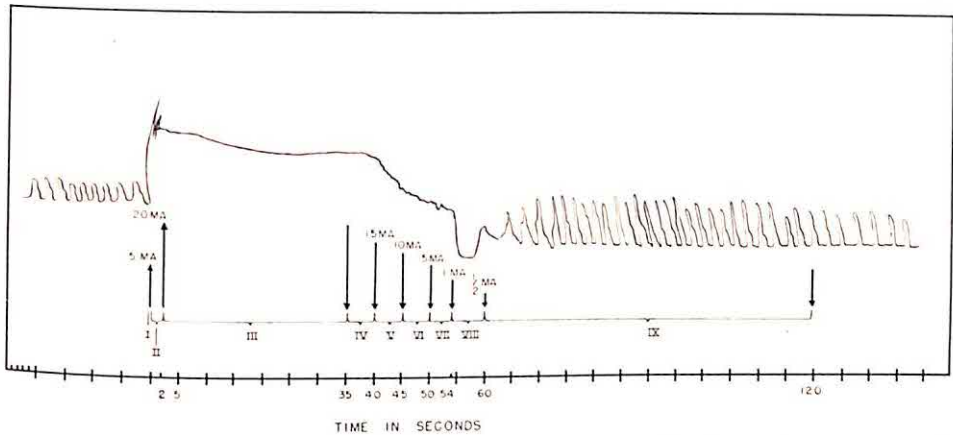


Fig. 89. Respirograph tracing of a patient undergoing combined convulsive-nonconvulsive electric treatment through frontal leads of two minutes' duration (54 seconds of tonic convulsive treatment, followed by 66 seconds of nonconvulsive stimulation). Arrows pointing upward denote turning on and raising of current, arrows pointing downward denote lowering of current. The Roman numerals refer to the steps of the procedure illustrated in the dial-face diagrams, Figs. 90-98 (Steps I-IX).

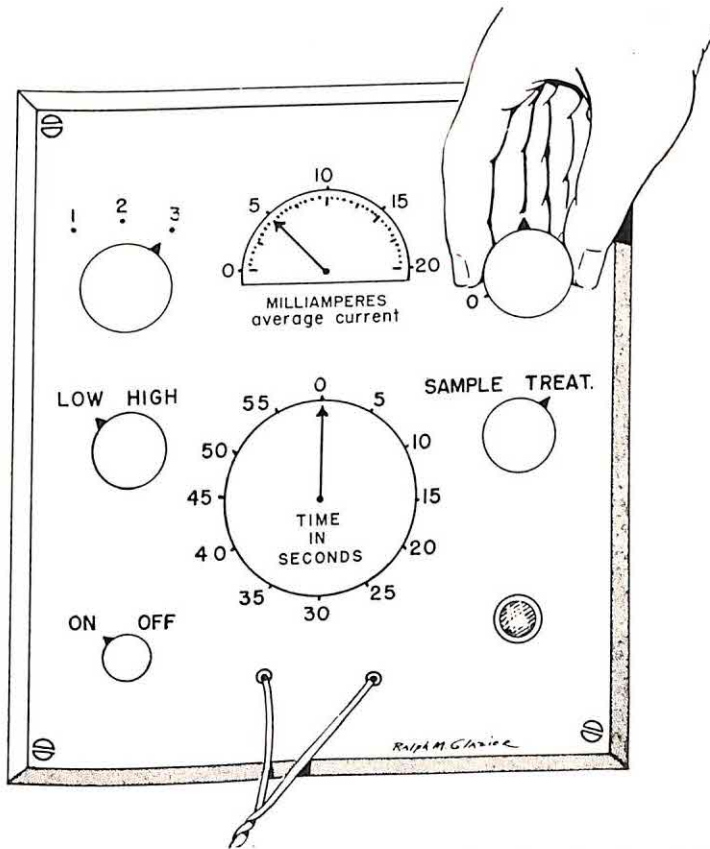


Fig. 90. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration. This and the following eight dial-face diagrams illustrate the treatment administered with the Reiter CW 46 electrostimulator. Step I. Set current control dial at average current of 5 MA.

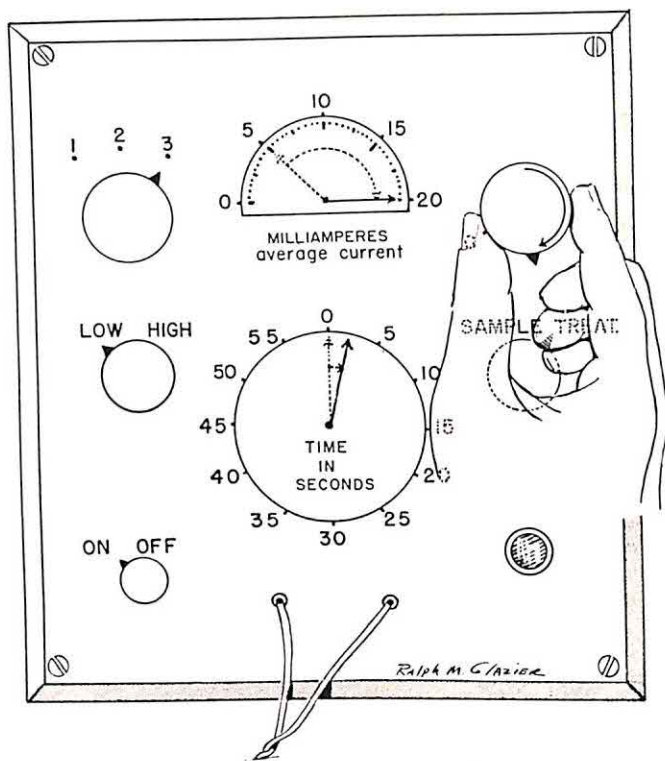


Fig. 91. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step II. Raise current to 20 MA within 2 seconds.

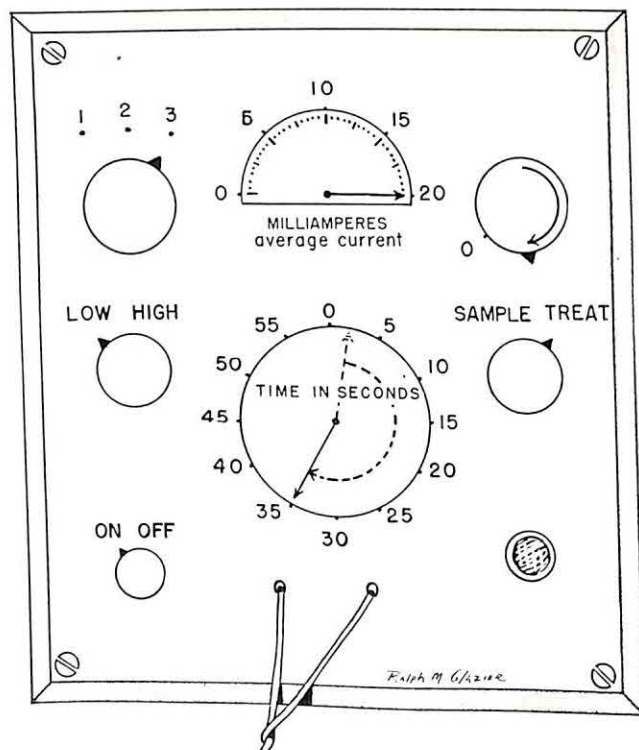


Fig. 92. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step III. Hold current at 20 MA for remainder of 35 seconds.

action becomes particularly dangerous when combined with postshock apnea, thus imposing the additional stress of anoxia upon the heart muscle and the impulse-conducting system. This combination of stress factors contributes serious risk of fatality. It is therefore important always to prevent apnea by all means available. If a patient treated with massive amounts of alternating current does not breathe immediately after the convulsion, artificial respiration should be instituted at once, without wait-

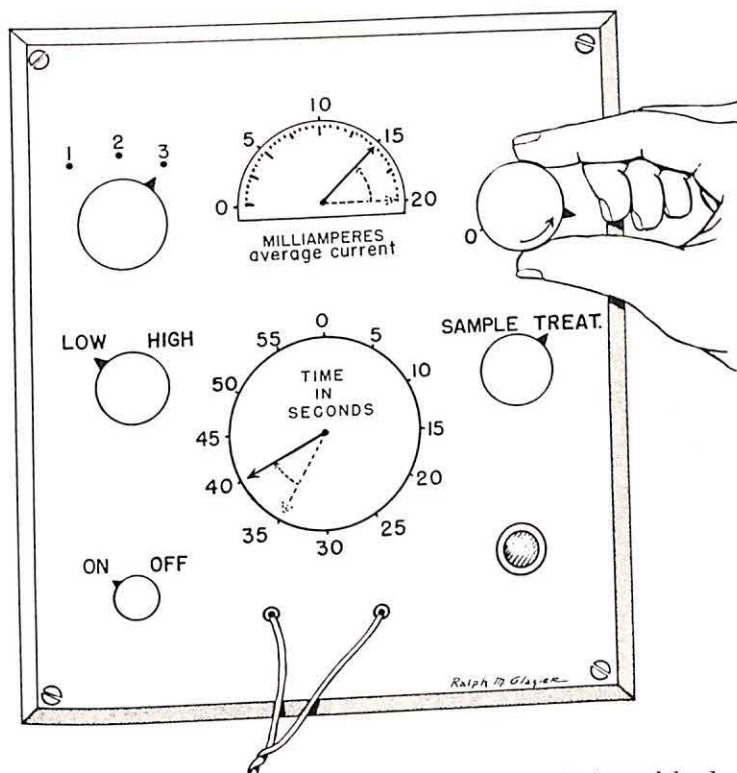


Fig. 93. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step IV. Reduce current gradually to 15 MA within 5 seconds.

ing for tentative spontaneous respirations to appear. A few lifting movements of the chest will usually suffice to initiate adequate spontaneous respiration. When unidirectional currents are used, shifting to nonconvulsive stimulation at the level of $\frac{1}{2}$ milliampere of average current (with spikes up to 70 milliamperes) will usually start adequate respiration promptly. In view of the great general advantages of postelectroshock stimulation at nonconvulsive levels, I always combine convulsive electroshock—if given by means of unidirectional currents—with a minimum of 60 seconds of nonconvulsive treatment. My standard technique will be described in the following section.

Combined Convulsive-Nonconvulsive Treatments

Combined Convulsive-Nonconvulsive Treatment through Frontal Leads of Two Minutes' Duration. This treatment is given with the Reiter CW 46 machine, through bifrontal leads. It lasts for two minutes; 54 seconds are allowed for treatment at convulsive levels, 66 seconds for treatment at

nonconvulsive levels. During the convulsive part of the treatment, the clonic phase is suppressed by continued stimulation at full strength of the current for the first 35 seconds. From then on the current is gradually lowered so that intrathoracic pressure is reduced while clonus and respiration are still inhibited, the lowering of the current blending gradually into the nonconvulsive phase at which point respiration is resumed. I have endeavored to time the treatment precisely, so that the first breath appears at 54 seconds—the usual maximum time span of the convulsion (see Chapter IX).

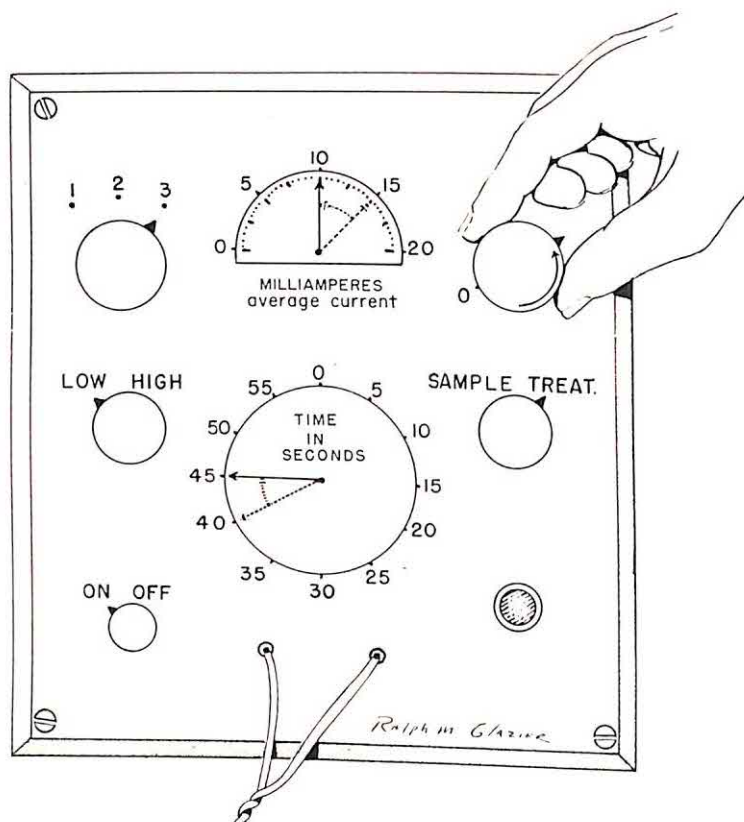


Fig. 94. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration. Step V. Reduce current gradually to 10 MA within 5 seconds.

The technique I have developed involves nine major steps. The accompanying illustrations demonstrate these steps, while Fig. 89 indicates the concomitant changes in the patient's respiration.

Step I. Set the machine at an average current of 5 milliamperes.* (See Figs. 89 and 90.)

Step II. Step on the foot switch and with a gentle even movement of the manual current control dial at the upper right corner of the control panel

* Spikes superimposed over the unidirectional current used in this machine are 62 to 140 times the average current: for example, an average current of $\frac{1}{2}$ milliamperes means a current with spikes up to 370 milliamperes; an average current of 5 milliamperes means a current with spikes up to 1670 milliamperes (see Chapter VIII).

raise the current up to 20 milliamperes within 2 seconds (Figs. 89 and 91).

Step III. Maintain the current at the level of 20 milliamperes of average current until the time clock indicates that 35 seconds have elapsed (Figs. 89 and 92).

After a very brief latency period, the patient will go immediately into tonic extension and will retain the tonic extension throughout this and the following periods of the convulsive phase. The latency period usually lasts only 1 to 2 seconds, rarely exceeding 8 seconds (latencies are longer

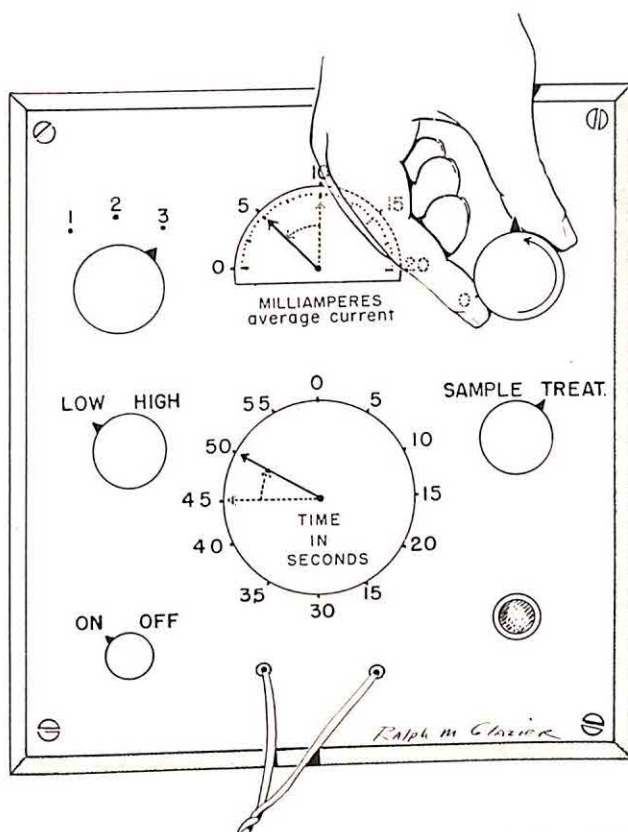


Fig. 95. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step VI. Reduce current gradually to 5 MA within 5 seconds.

when less current is used). During this brief latency period only one or a few tetanic contractions occur, the height of the tetanic contraction becoming almost instantly fixed in the tonic phase.

The extensor spasm of the tonic phase thus initiated involves neck, trunk, legs, and feet. The arms are usually flexed at the elbows, always flexed at the wrist in Wartenberg position. Occasionally patients will show extension—sometimes only a transitory extensor thrust—of the elbow of one arm in this phase of the treatment; it is usually the one opposite the positive pole.

Step IV. At 35 seconds, begin reducing the current gradually so that it is decreased to 15 milliamperes at 40 seconds. The patient will still be in tonic extension at that time (Figs. 89 and 93).

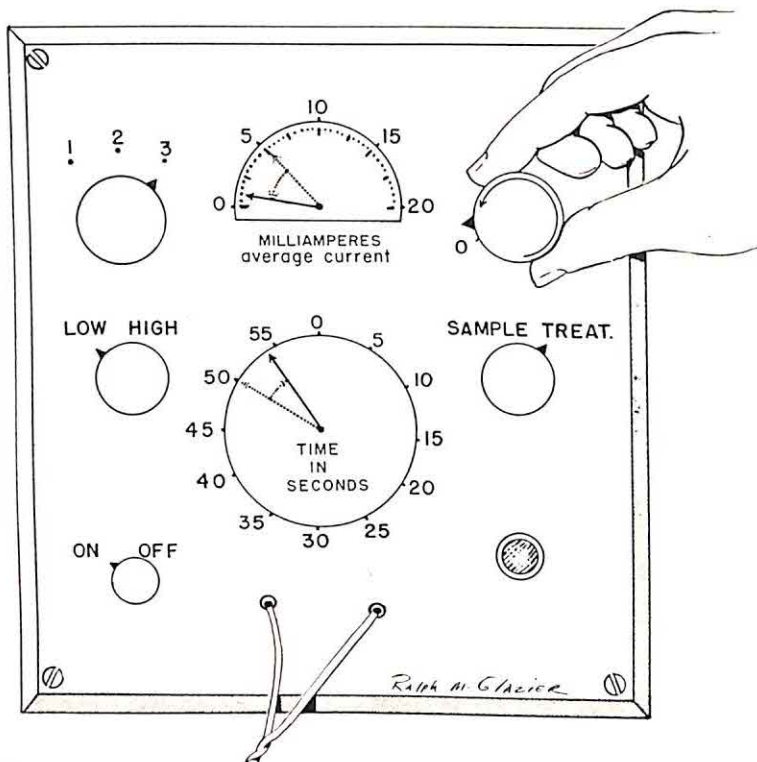


Fig. 96. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step VII. Reduce current gradually (within 4 seconds) to 1 MA.

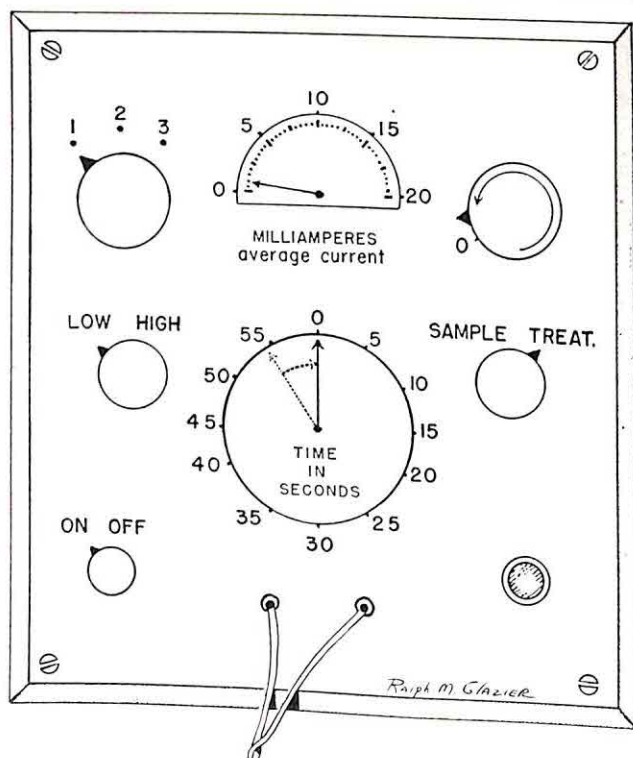


Fig. 97. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step VIII. Hold current at 1 MA for 6 seconds, while changing wave pattern from No. 3 to No. 1.

Step V. At 40 seconds, slowly reduce current from 15 to 10 milliamperes over a 5-second period, so that it will be 10 milliamperes at 45 seconds. During this phase intrathoracic pressure will lessen (see Fig. 89) and thus the Valsalva phenomenon will be released gradually. Also a slight amount of clonus may come through at this phase of treatment, although frequently the tonic extension is maintained throughout this period (Figs. 89 and 94).

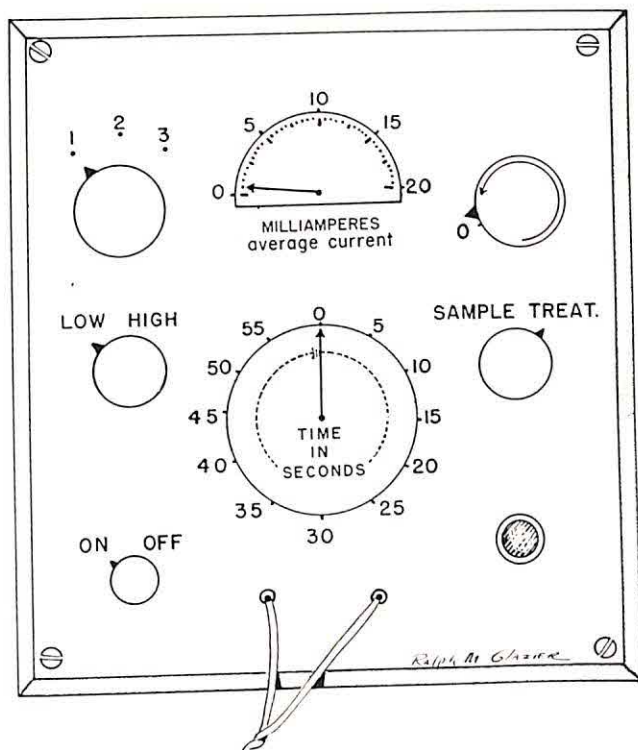


Fig. 98. Combined convulsive-nonconvulsive treatment through frontal leads of two minutes' duration, Step IX. Reduce current to $\frac{1}{2}$ MA and hold there for one full minute.

Step VI. At 45 seconds, reduce the current gradually so that it will be 5 milliamperes at 50 seconds. If the convulsion has not yet ended, a slight amount of clonus will be apparent in this phase. Intrathoracic pressure will continue to decline gradually. (See Figs. 89 and 95.)

Step VII. At 50 seconds, reduce the current from 5 milliamperes to 1 milliampere over a 4-second period. At this point—i.e., at 54 seconds—the patient will become limp, and if the time schedule was carried out correctly and the patient does not fall into the group having an excessively low rheobase, he will begin breathing immediately. (See Figs. 89 and 96.)

Step VIII. At 54 seconds, the current should be maintained at 1 milliampere for 6 more seconds, but the wave pattern must be changed from No. 3 to No. 1 (see upper left corner of Fig. 97). Let the current continue to run at 1 milliampere until the clock reaches 60 seconds. If breathing is satisfactory at that point, the current may be allowed to run longer at this level, namely throughout the full additional 60 seconds of nonconvulsive stimulation.

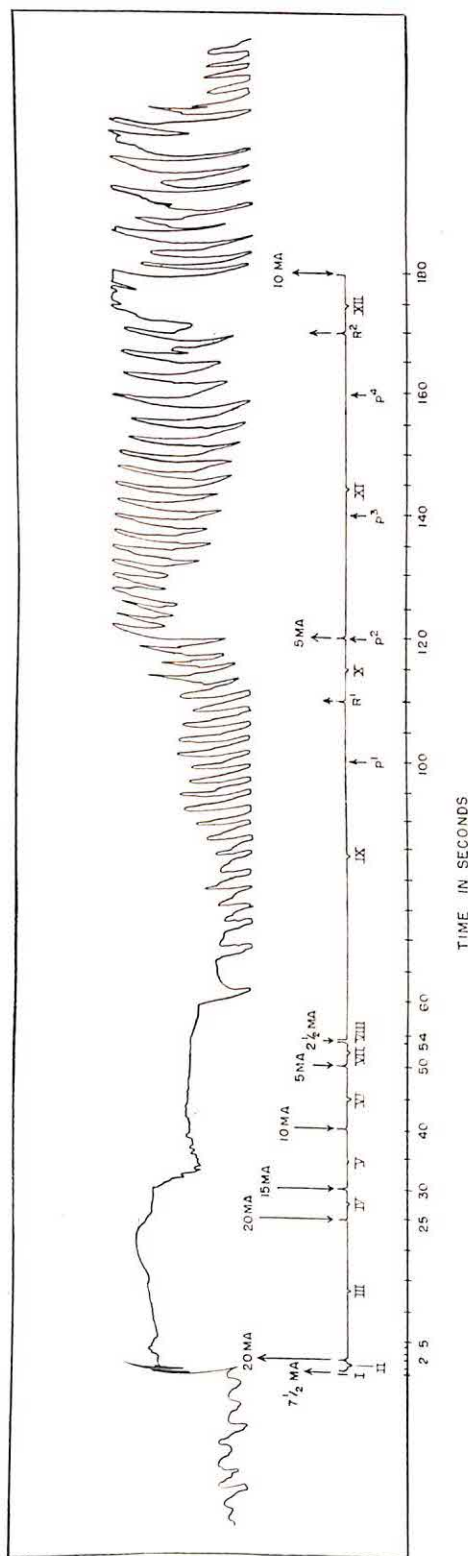


Fig. 99. Respirograph tracing of a patient undergoing combined convulsive-nonconvulsive electric treatment through temporoparietal leads of three minutes' duration (54 seconds of tonic convulsive treatment followed by 126 seconds of nonconvulsive stimulation). The strength of the stimulating current in MA is indicated; the accompanying arrows (above the horizontal subdivided line marked by Roman numerals) pointing upward denote that the current has been turned on or raised, pointing downward denote that the current has been lowered. The arrows below this line indicate changes in polarity. The Roman numerals refer to the steps of the procedure illustrated in the dial-face diagrams (Figs. 100-113, Steps I-XII).

P¹—First change in polarity 100 seconds after beginning of treatment.

P²—Second change in polarity 120 seconds after beginning of treatment.

P³—Third change in polarity 140 seconds after beginning of treatment.

P⁴—Fourth change in polarity 160 seconds after beginning of treatment.

R¹—Beginning of raising of current 110 seconds after beginning of treatment, reaching 5 MA 120 seconds after beginning of treatment.

R²—Beginning of raising of current 170 seconds after beginning of treatment, reaching 10 MA 10 seconds later, i.e., at end of treatment.

Step IX. If respiration is not satisfactory at the sixtieth second of the treatment, i.e., after 6 seconds of nonconvulsive stimulation at the level of 1 milliampere, the optimal level for facilitation and stimulation of respiration must be found, and the following 60 seconds of nonconvulsive stimulation must be administered at that level. This optimal level is just below the level at which respiration is still inhibited, usually with some laryngeal stridor, and just above the level at which it remains too shallow and slug-

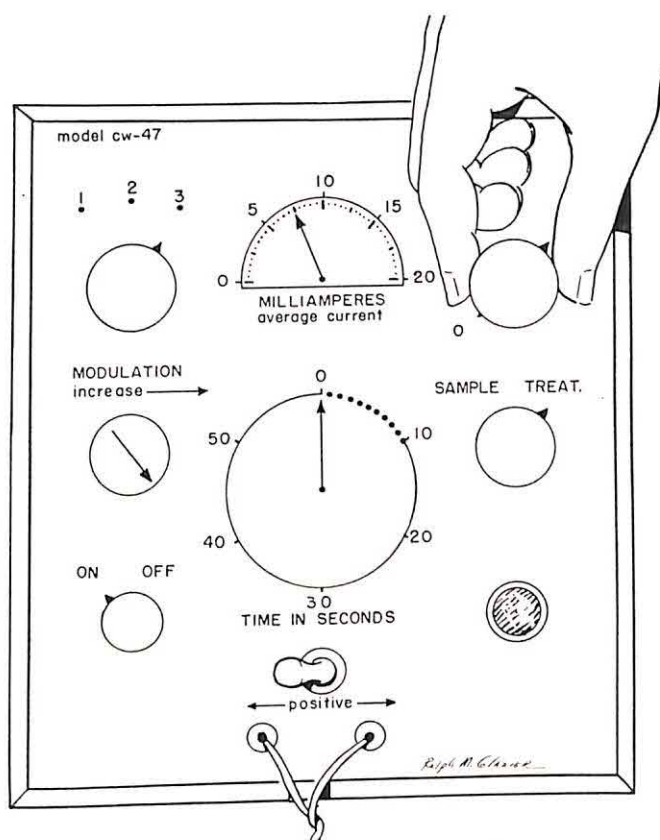


Fig. 100. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration. This and the following eleven dial-face diagrams illustrate the treatment administered with the Reiter CW 47 electro-stimulator. Step I. Set current control dial at average current of $7\frac{1}{2}$ MA. Turn on current.

gish. Usually this optimal level will be found at $\frac{1}{2}$ milliampere, and in this case stimulation should be continued at that level for another full minute (see Figs. 89 and 98). In some cases, however, this optimal level may be anywhere between $\frac{1}{2}$ and 2 milliamperes, and the physician should be ready to make an appropriate readjustment of the current level at any time during the ensuing minute of nonconvulsive stimulation.

If in spite of this technique there should be a period of apnea (which must of course immediately be dealt with by artificial respiration), we must assume it is because the patient falls in the group of persons who have an excessively low rheobase. In such cases it is best in subsequent treatments to raise the current in Step II to only 15 milliamperes, and to

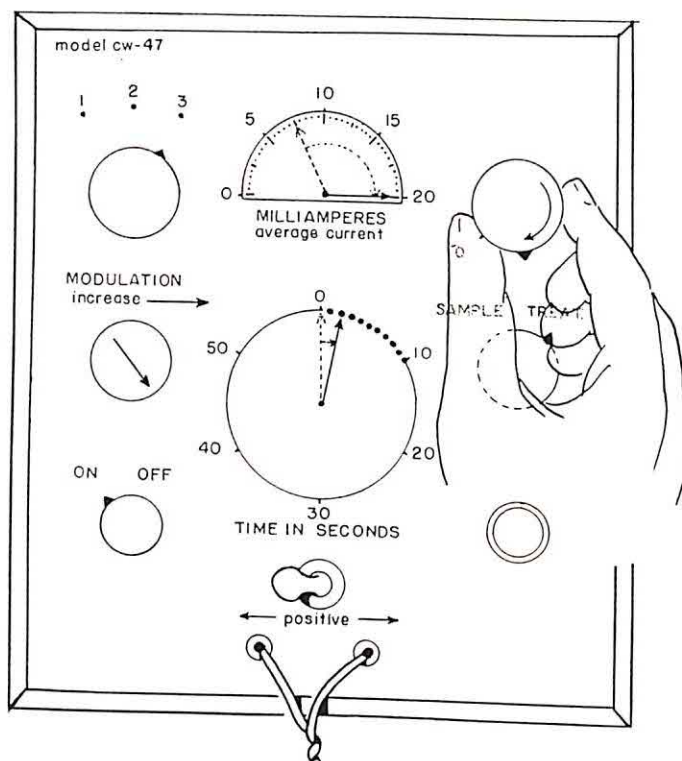


Fig. 101. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step II. Raise current to 20 MA within 2 seconds.

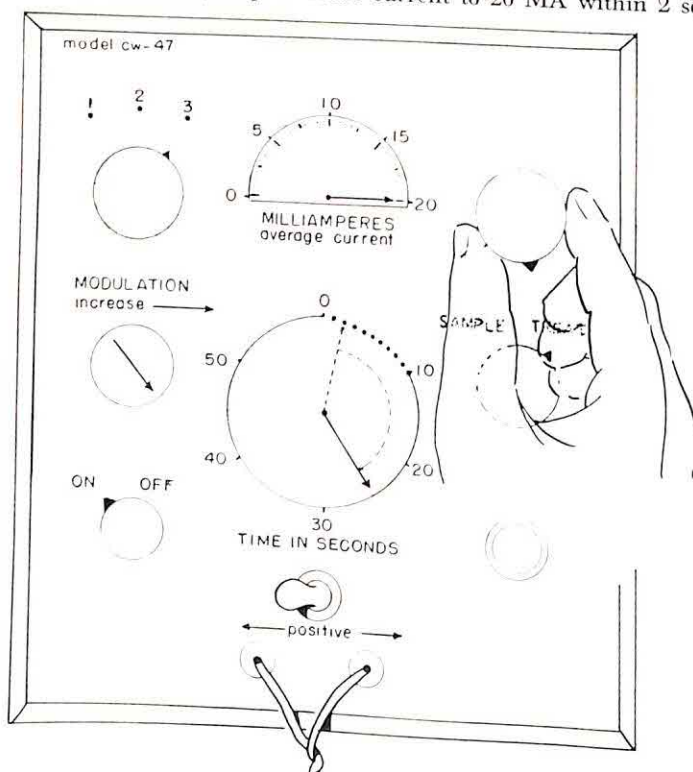


Fig. 102. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step III. Hold current at 20 MA for remainder of 25 seconds.

carry out the other steps as before, so that 10 milliamperes will be reached at 40 seconds, 5 milliamperes at 45 seconds, 1 milliampere at 50 seconds, and $\frac{1}{2}$ milliampere at 54 seconds. Nonconvulsive stimulation may then be continued at $\frac{1}{2}$ milliampere for the remainder of the total time span of two minutes. One may also start reducing the current to 10 milliamperes after only 10 to 25 seconds. It is not necessary to reach the 5 milliamperes level before the 45 second mark. Recently I treated such a patient by raising the current to only 15 milliamperes at Step II and reducing it to 10 milliamperes as soon as the extensor spasm was definitely established (10 seconds), then gradually decreasing it further until 5 milliamperes was reached at 45 seconds.

The disadvantage of this technique, however, is that it does not produce as much vasodilatation as the full regimen outlined above in the preceding nine steps. In a patient with coronary heart disease whom I treated with an earlier return to low current levels, prolonged and alarming

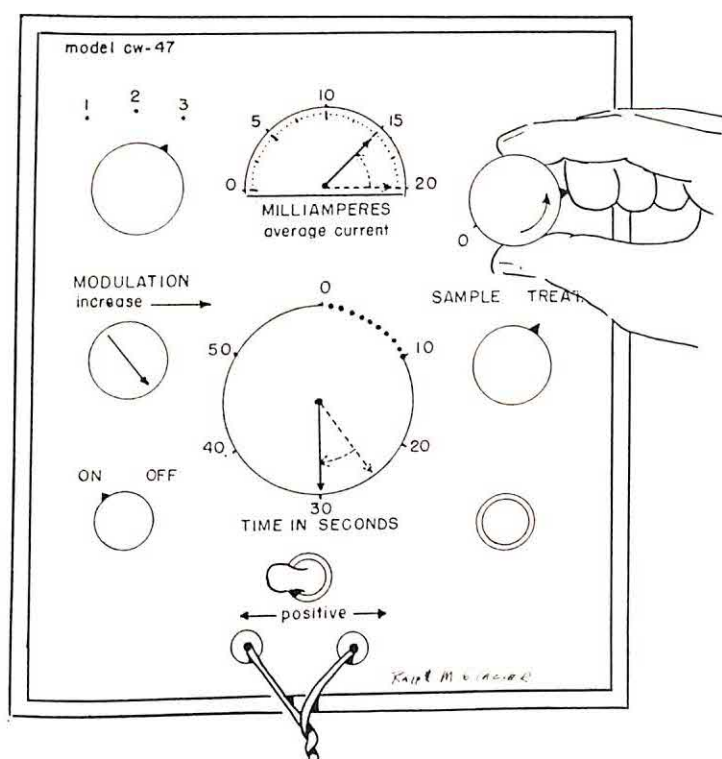


Fig. 103. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step IV. Reduce current gradually to 15 MA within 5 seconds.

peripheral vasoconstriction, dyspnea, and hyperpnea—the respirations being far more rapid and labored than the usual hyperpnea after electroshock treatment—(associated with marked pilomotor erection or gooseflesh) continued for an alarmingly long period after the convulsive seizure. When I subsequently treated this patient with the full current maintained for the full periods, peripheral vasodilatation manifested itself from the 35th second of the treatment onward and was maintained throughout the postconvulsive period. Gooseflesh, as well as excessive dyspnea and hy-

perpnea, was absent. This excellent tolerance of this type of electroshock treatment was maintained throughout the remaining six treatments of the series, enabling this patient to make an uneventful recovery without exacerbation of her coronary heart disease.

The treatment described above is the standard treatment I employ for depressions that are either entirely free of epinephrine-precipitable anxiety or show only slight or moderate anxious reactions to epinephrine—in other

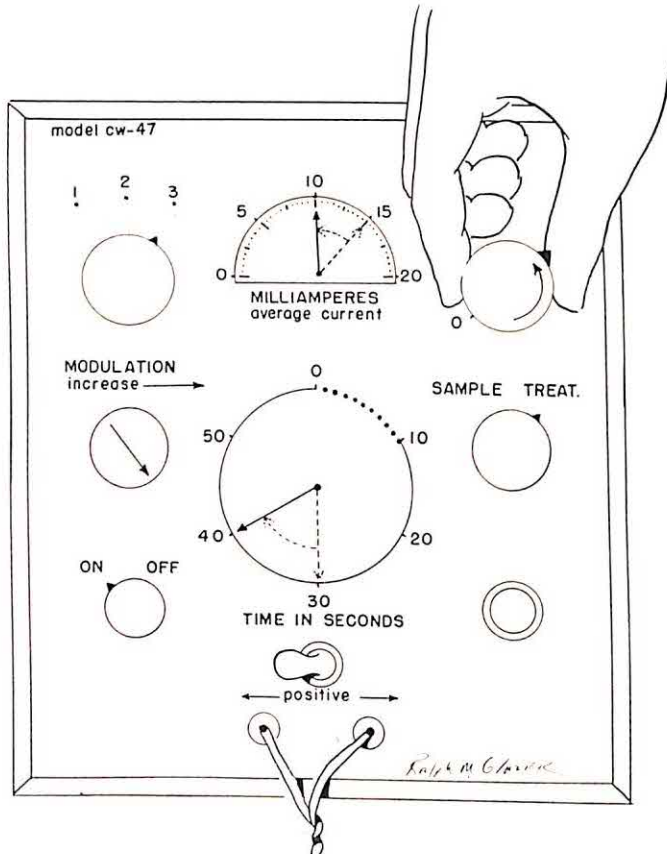


Fig. 104. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step V. Reduce current gradually to 10 MA within 10 seconds.

words, for the classic depressions and for those only slightly complicated by anxiety.

This treatment has a marked antidepressant effect except in the most severe involutional depressions in which the amount of electric stimulation delivered by this method is sometimes too small, as is evidenced by the fact that it cannot produce extensor thrust of both arms in these patients. Neither does this method help mania, which generally requires either all-out convulsive treatment or purely nonconvulsive treatment depending on the autonomic response pattern. Depressions other than the most severe involutional ones do not require stimulation sufficiently strong to produce extensor thrust of the arms—this is especially true of the ordinary midlife depressions which are the ones that respond best to this method.

In a few rare instances this treatment also fails to produce extension of the legs, but instead the legs are flexed at the hips and knees, as described in Chapter IX. Together with the flexion of the arms, this results in a position referred to as "jumping jack position" (von Braunmuehl,¹²³ Kalinowsky and Hoch,¹³⁰ page 113). This is obviously due to the fact that the treatment has merely stimulated the cortex and has failed to fire off the subcortical structures. This may sometimes be due to sedation, especially

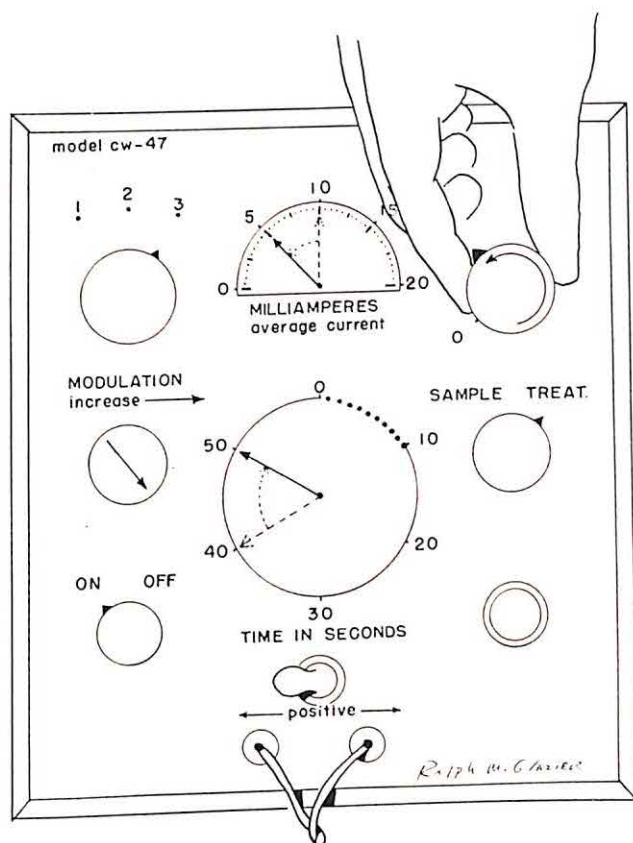


Fig. 105. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step VI. Reduce current gradually to 5 MA within 10 seconds.

with dilantin, which apparently prevents the firing off of the subcortical structures, while the cortical excitability remains less affected. Otherwise it must be due to an unusually high rheobase. Temporary flexion of the legs is more frequent, extension of the legs coming through at the end of the convulsive phase of the treatment. If no extension results at all, even if the peak current has been maintained for a full 35 seconds or more, the antidepressant effect of the treatment will remain only moderate and will be insufficient for severe depressions. In such cases more powerful currents than those used in this form of treatment will have to be resorted to, i.e. either treatment with the same Reiter machine on the high scale or treatment with one of the standard alternating current machines.

Combined Convulsive-Nonconvulsive Treatment of Three Minutes' Duration through Temporoparietal Leads. This treatment is given with the

Reiter CW 47 machine through bi-temporoparietal leads. It lasts for three minutes; 54 seconds is allowed for the treatment at convulsive levels and 126 seconds for treatment at nonconvulsive levels. During the convulsive part of the treatment, the clonic phase is suppressed by continued stimulation for the first 25 seconds. The flow of the current at full strength is restricted to 25 seconds because this treatment exerts a potentially longer lasting inhibitory effect on respiration, presumably because of the further

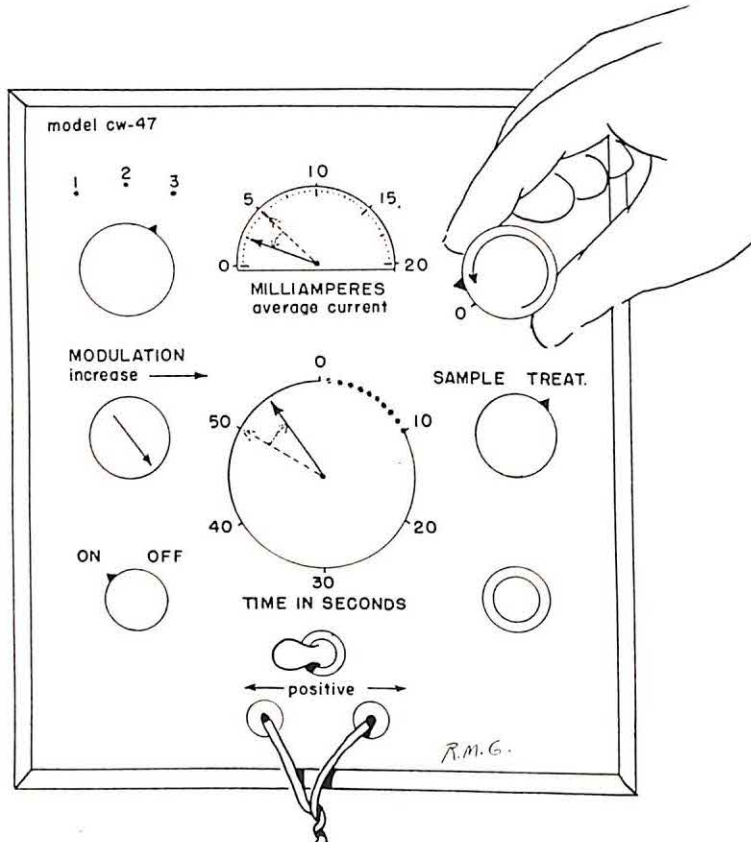


Fig. 106. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step VII. Reduce current gradually (within 4 seconds) to $2\frac{1}{2}$ MA.

posterior placement of the electrodes. However, 25 seconds of the full strength of the current is easily tolerated without causing significant post-shock apnea. From 25 seconds on, the current is gradually lowered so that a minimum of clonus is allowed to come through gradually while respiration is still inhibited, the lowering of the current blending gradually into the non-convulsive phase at which point the respiration is resumed. Because of the different ratio of average current and peak-to-peak current in the non-convulsive range of currents obtainable from this machine, the ideal range of respiration-stimulating nonconvulsive current, approximately corresponding to the action of $\frac{1}{2}$ to $\frac{3}{4}$ milliamperes (with spikes up to 70 to 100 milliamperes) on the CW 46 machine, is a current measuring $2\frac{1}{2}$ milliamperes with spikes up to 98 milliamperes on this CW 47 machine. In the convulsive range, currents obtainable from the CW 47

machine are identical with those of the convulsive range on the CW 46 machine. On Reiter's new combined model CW 47 B dosages in the nonconvulsive range are similar to those for the CW 46 machine (see Figs. 97 and 98).

The following twelve specific steps are recommended for administration of this form of treatment with the CW 47 machine:

Step I. Set the machine at an average current of $7\frac{1}{2}$ milliamperes. The current selector at the upper left-hand corner of the dial face should be in

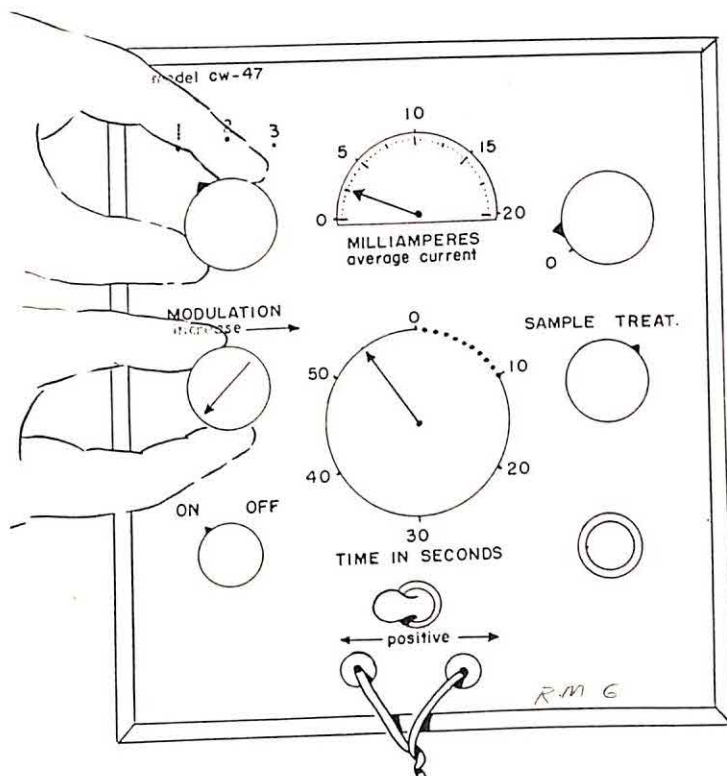


Fig. 107. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step VIII. Change current type from No. 3 to No. 1 and change modulation to maximally slow by turning modulation knob counterclockwise as far as it will go (to 7 o'clock).

position 3 where current suitable for convulsive stimulation, identical with the current of the CW 46 machine, is obtained. (See Chapter VIII, Figs. 7 and 8.) Maximally fast modulation should be employed with the modulation knob turned as far clockwise as it will go (5 o'clock). Turn on the treatment switch of the machine. (Figs. 99 and 100.)

Step II. Immediately after turning on the machine, raise the current to 20 milliamperes within 2 seconds, by means of the current control dial in the upper right hand corner (Figs. 99 and 101).

Step III. Maintain the current at the level of 20 milliamperes of average current for the remainder of 25 seconds (Figs. 99 and 102).

Step IV. When 25 seconds have elapsed, gradually reduce the current over a 5-second period, so that it is decreased to 15 milliamperes at 30 seconds (Figs. 99 and 103).

Step V. At 30 seconds, slowly reduce the current from 15 to 10 milliamperes over a ten-second period, so that 10 milliamperes of current will be flowing at 40 seconds. During this phase, a partial release of the Valsalva phenomenon as well as the emergence of a slight amount of clonic activity will usually be noted (Figs. 99 and 104).

Step VI. At 40 seconds, reduce the current gradually over a 10-second period so that it will be 5 milliamperes at 50 seconds (Figs. 99 and 105).

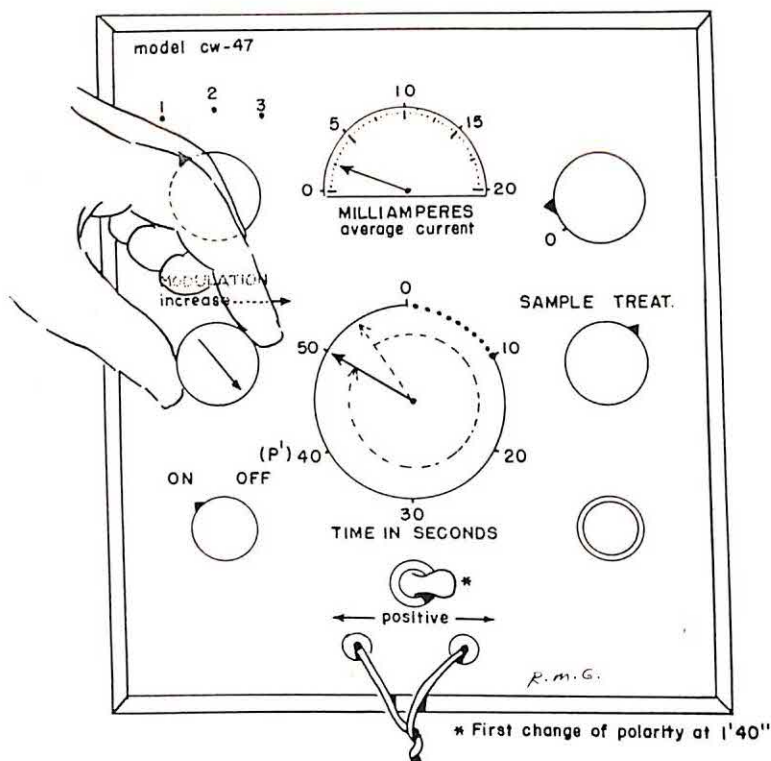


Fig. 108. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step IX. Restore modulation to maximally fast by turning modulation knob clockwise as far as it will go (to 5 o'clock). Keep current at $2\frac{1}{2}$ MA for 56 seconds. Carry out first change of polarity at the 40 second mark of the second minute of treatment at the point indicated "P1" (1 minute, 40 seconds after beginning of treatment).

Step VII. At 50 seconds, reduce the current from 5 milliamperes to $2\frac{1}{2}$ milliamperes over a 4-second period, so that it is decreased to $2\frac{1}{2}$ milliamperes at the 54 second mark (Figs. 99 and 106).

Step VIII. As soon as the current has been reduced to an average current of $2\frac{1}{2}$ milliamperes at 54 seconds, the current selector should be turned to No. 1 position from which an exponential condenser discharge type of current is obtained, which is eminently suitable for nonconvulsive stimulation (see Chapter VIII, Figs. 9 and 10). At the same time, the modulation knob should be turned to the maximally slow position by turning it counterclockwise to 7 o'clock (Fig. 107). This slowing of modulation frequency (see Chapter VIII, Fig. 9 B as compared with Fig. 9 A) is particularly conducive to the stimulation of respiratory activity. At this time, or shortly

thereafter, the patient will start breathing (Fig. 99). If the patient does not start breathing within 6 seconds after the decrease of the current, the current should be lowered to 2 milliamperes; if this does not initiate respiration, the current should rapidly be raised to 10, and immediately and equally rapidly be lowered back to 2 milliamperes; this maneuver may be repeated two to three times, aided by a few lifting movements of the chest or other forms of artificial respiration. In the rare event that spontaneous respiration may still not be forthcoming, the current should be turned off and artificial respiration continued. Throughout the course of adminis-

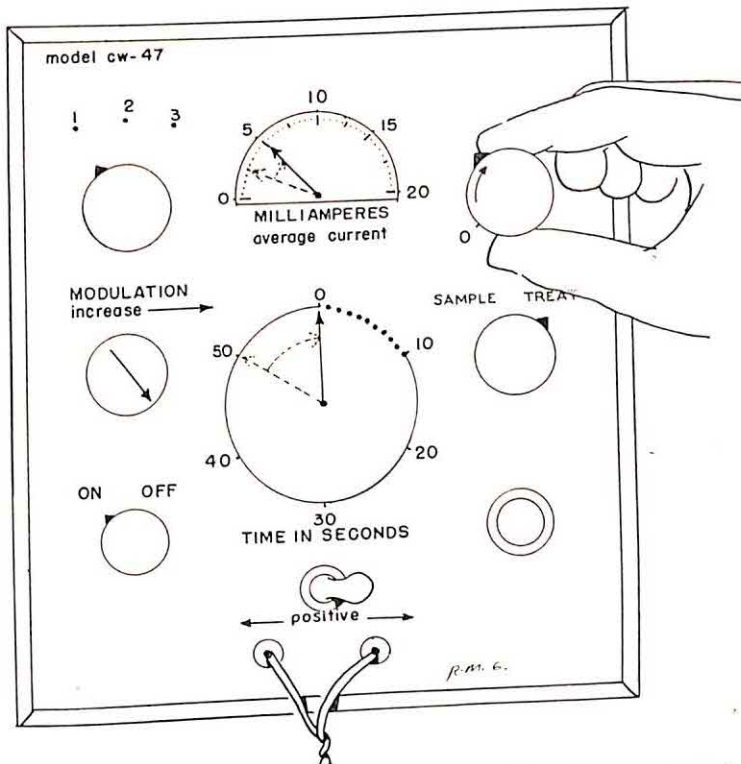


Fig. 109. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step X. Raise current to 5 MA within 10 seconds.

tration of these measures, care must be taken that the air passage be unobstructed. Suction and tracheal tube must be available.

Step IX. As soon as respiration is adequate, the modulation knob should be turned back to the maximally fast position while nonconvulsive stimulation with the current selector on No. 1 position at an average current of $2\frac{1}{2}$ milliamperes is continued for a total of 56 seconds, i.e., until the time clock reaches the 50 second mark of the second minute.

When the current reaches the 40 second mark of the second minute (100 seconds after the beginning of the treatment), polarity should be reversed for the first time by turning the polarity knob to the right (Figs. 99 and 108).

Step X. At 1 minute and 50 seconds, provided the respirations are deep and adequate, the patient is ready to tolerate larger amounts of current

without suffering arrest of respiration or responding with a second convulsion, provided the same type of exponential condenser discharge type of current is used with which the period of nonconvulsive stimulation was initiated. The current selector knob should therefore remain at the No. 1 position throughout the nonconvulsive phase of the treatment. Also, the modulation knob must remain in the maximally fast position from this point on, unless the patient should develop respiratory distress, at which time the modulation frequency should again be decreased. However, this rarely occurs with this form of treatment.

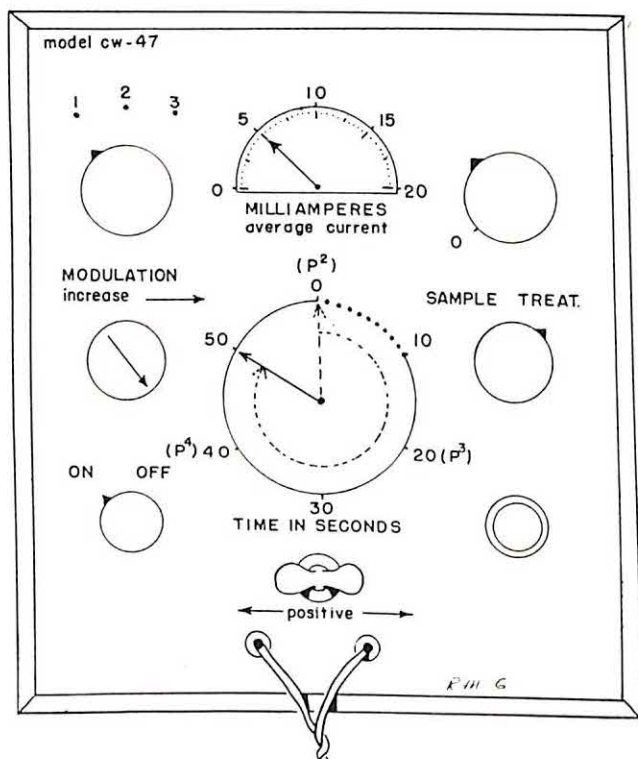


Fig. 110. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step XI. Hold current at 5 MA for 50 seconds after 20 seconds and after 40 seconds (at points marked P³ and P⁴).

At 1 minute and 50 seconds, the current should be gently and slowly raised, preferably only during the expiratory phases of respiration until the current reaches an average level of 5 milliamperes (with spikes up to 160 milliamperes) at the 2-minute mark. (Fig. 109.) Even if this is done carefully and gently, there will be some decrease in the depth of expirations and an increase in intrathoracic pressure as a result of the current increase (Fig. 99).

Step XI. As soon as the current reaches the 5 milliamperes mark, polarity should be reversed to its original position and the stimulation continued at that level for a total of 50 seconds. Polarity should be changed twice during the third minute, once at 2 minutes and 20 seconds, and again at 2 minutes and 40 seconds (Fig. 110). Respiration will gradually

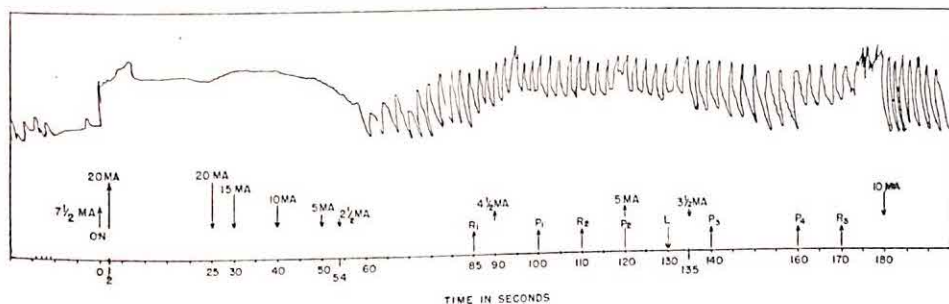


Fig. 111. Respirograph tracing of a patient undergoing combined convulsive-nonconvulsive electric treatment through temporoparietal leads of three minutes' duration (54 seconds of tonic convulsive treatment followed by 126 seconds of nonconvulsive stimulation). The upper row of arrows denotes turning on and raising of current (points of arrows upward), or lowering and turning off of current (points of arrows downward), the milliamperage reached at those points being indicated above the arrow. The lower row of arrows indicates the beginning of the raising or lowering maneuvers of the current, as well as the changes in polarity.

R¹—First raising of current, 85 seconds after beginning of treatment.

P¹—First change in polarity, at 100 seconds.

R²—Beginning of second raising of current, at 110 seconds.

P²—Second change of polarity, at 120 seconds.

L—Beginning of lowering of current, at 130 seconds.

P³—Third change in polarity, at 140 seconds.

P⁴—Fourth change in polarity, at 160 seconds.

R³—Third raising of current, at 170 seconds, reaching 10 MA by the end of the treatment.

Note limitation of expirations and increase in intrathoracic pressure after the current was raised from 2½ to 4½ MA between the 85th and the 90th seconds of treatment, promptly relieved as soon as the current was reduced to 3½ MA after 135 seconds of treatment.

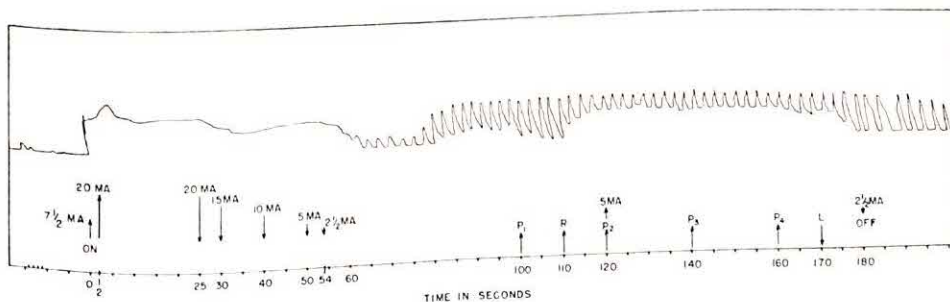


Fig. 112. Respirograph tracing of a patient undergoing combined convulsive-nonconvulsive electric treatment through temporoparietal leads of three minutes' duration (54 seconds of tonic convulsive treatment followed by 126 seconds of nonconvulsive stimulation). Upper row of arrows denotes accomplished changes in current level; lower row of arrows denotes beginning of raising or lowering of current as well as changes in polarity.

P¹—First change in polarity, at 100 seconds.

R—Beginning of raising of current, at 110 seconds.

P²—Second change in polarity, at 120 seconds.

P³—Third change in polarity, at 140 seconds.

P⁴—Fourth change in polarity, at 160 seconds.

L—Beginning of lowering of current at 170 seconds, resulting in lowering to 2½ MA at time of completion of treatment.

Note reduction in depth of expirations after the current was raised (at 110 seconds), maintained until after it was lowered upon completion of 170 seconds of treatment.

return to its former depth of expiration and the intrathoracic pressure will decline (Fig. 99). If considerable relief does not ensue within 15 to 20 seconds, the current must be lowered. This was done in a patient whose expirations became restricted from the 90th second of treatment onward, that is, after the current level had been raised from $2\frac{1}{2}$ to $4\frac{1}{2}$ milliamperes, and subsequently to 5 milliamperes. When subsequently no tendency to adaptation and reexpansion of expirations took place for 45 seconds, it was decided to lower the current to $3\frac{1}{2}$ milliamperes, whereupon expira-

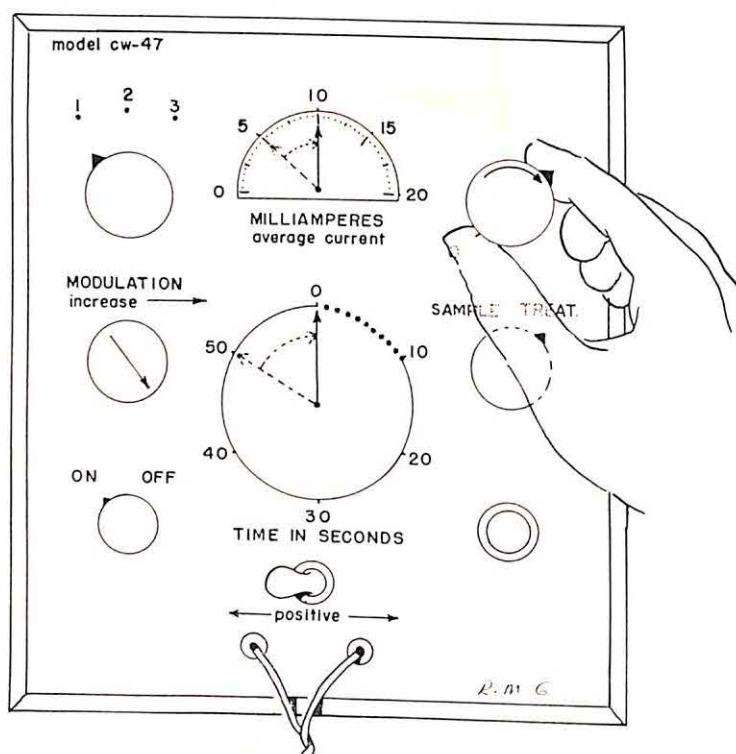


Fig. 113. Combined convulsive-nonconvulsive treatment through temporoparietal leads of three minutes' duration, Step XII. Raise current to 10 MA within 10 seconds, then shut off current.

tions promptly reexpanded (Fig. 111). The greater sensitivity of this patient to electric stimulation (lower rheobase) is also illustrated by the fact that when the current was raised to 10 milliamperes in the "end spurt" of the final 10 seconds of treatment, myoclonic contractions were elicited at that level (Fig. 111), while normally they are not (Fig. 99).

Expirations will usually not reexpand spontaneously unless the current is lowered. It is important to realize that, and to recognize that the reduction of depth of expirations constitutes a danger sign, which, if unheeded, may lead to cyanosis and anoxia. In general, this limitation of expirations can be tolerated for one minute without apparent cyanosis. Close attention to the respirograph tracing during the course of treatment will warn the therapist of this approaching danger in time for him to avert such a crisis.

Figure 112 shows the respirograph record of a patient who, when the

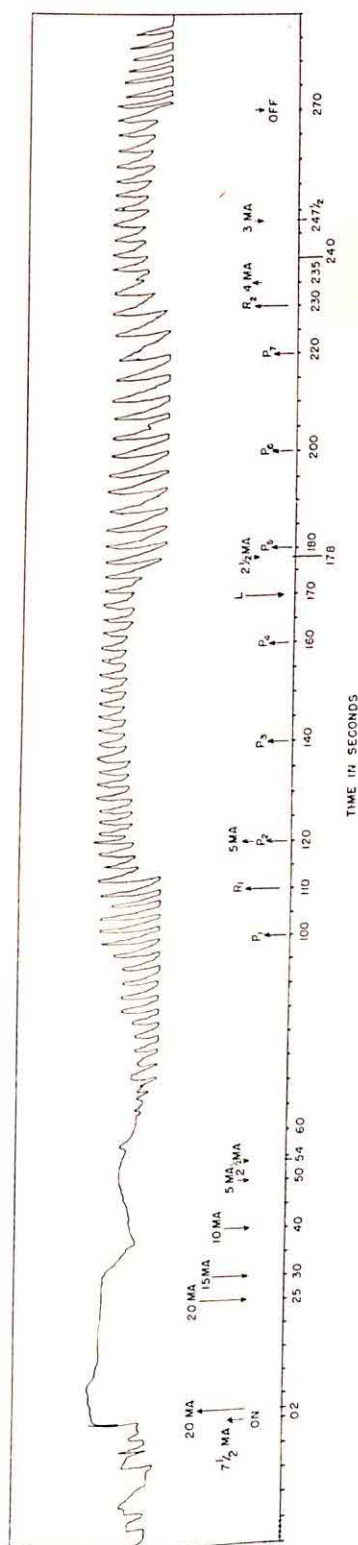


Fig. 114. Respirograph tracing of a patient undergoing combined convulsive-nonconvulsive electric treatment of 4½ minutes' duration (54 seconds of tonic convulsive treatment followed by 216 seconds of nonconvulsive stimulation). Levels of current indicated in milliamperes, with arrows indicating how this milliamperage was obtained. Arrows pointing upward denote that the current value indicated was obtained by raising of the current, arrows pointing downward denote that the milliamperage indicated has been obtained by lowering of the current. P1 to P7—Indicate seven changes in polarity 20 seconds apart, the first one at 100 seconds, the last one at 220 seconds.
 R1—Beginning of first raising of current at 110 seconds, reaching 5 MA at 120 seconds.
 R2—Second raising of current at 230 seconds reaching 4 MA at 235 seconds.
 L—Lowering of current at 170 seconds reaching 2½ MA at 178 seconds.

Note limitation of expirations when current was raised to 5 MA from the 120th to the 170th second of treatment, and similarly but proportionally less marked limitation of expirations when current was raised to only 3 MA during the last 40 seconds of the treatment. Note normal depth of expirations during the intervening 52 seconds (from 178 to 230 seconds) when the current was at an average level of 2½ MA.

Reexpansion of depth of expirations was prompt as soon as current was lowered during treatment or turned off at end of treatment.

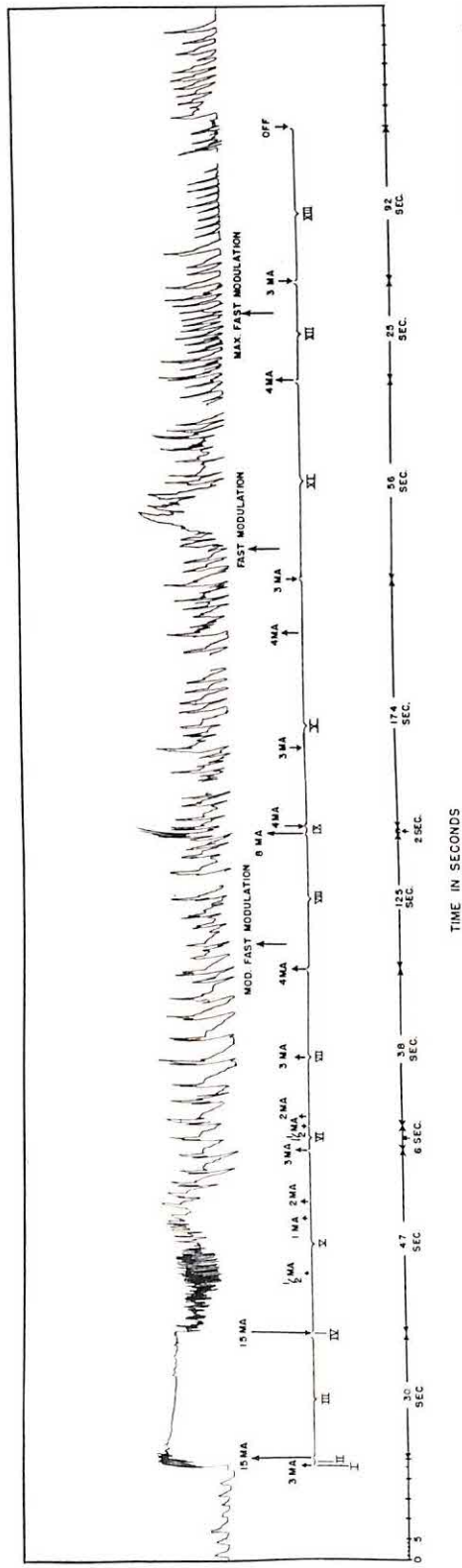


Fig. 115. Respirograph tracing of a patient undergoing electric coma treatment of 9 minutes and 57 seconds duration: a convulsive stimulus of 32 seconds' duration followed by an interval of 15 seconds, resulting in a partly modified convulsion, is followed by nonconvulsive stimulation of 9 minutes and 10 seconds duration. Levels of electric current are indicated in milliamperes, with arrows indicating how each level was attained; arrows pointing upward denote that the current value given was obtained by raising of the current, arrows pointing downward denote that the milliamperage indicated was obtained by lowering of the current. Changes in modulation frequency are likewise indicated. The Roman numerals and corresponding brackets refer to the steps of the procedure illustrated in the dial-face diagrams (Figs. 116-128, Steps I-XIII); their duration is indicated at the lower margin of the graph.

current was raised to 5 milliamperes as is usual at the end of the second minute of treatment, responded with a reduction of expirations to one-half their former depth, without other change in rhythm or quality of respiration. Her general condition and appearance remained good until 60 seconds later when slight cyanosis became evident. The current was therefore instantly lowered, expirations reexpanding as soon as the current was reduced to 3 milliamperes, and becoming fully normal at $2\frac{1}{2}$ milliamperes, at which level the treatment was terminated at the end of the third minute.

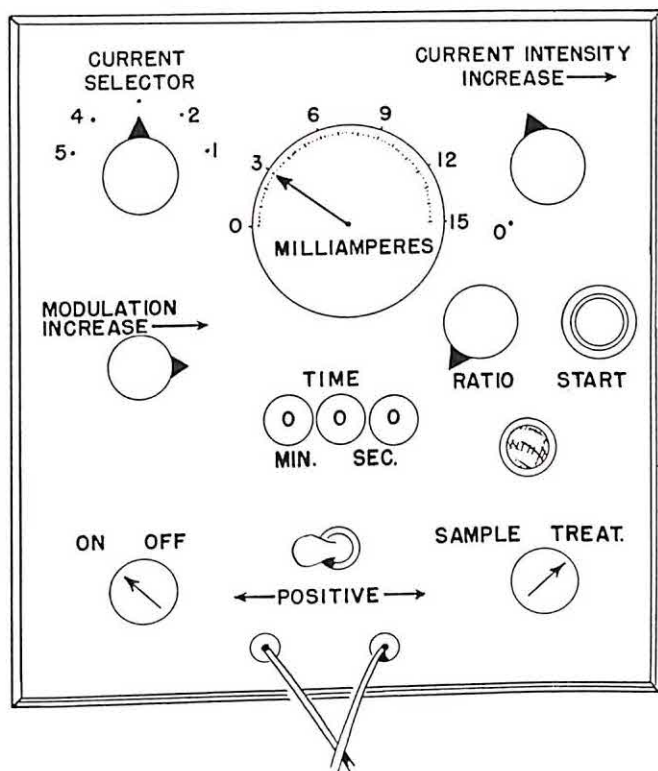


Fig. 116. Electric coma. This and the following twelve dial-face diagrams illustrate the treatment administered with the Reiter RC 47 electrostimulator. Step I. Set current control dial at average current of 3 MA. Turn on current.

Step XII. At 2 minutes and 50 seconds, the current should be gradually increased to an average current of 10 milliamperes (with spikes up to 252 milliamperes) for the remaining 10 seconds of the treatment ("end spurt"), at which time the current should be shut off (Fig. 113). Respiration will usually be inhibited as soon as the current is raised, beginning at an average level of $7\frac{1}{2}$ milliamperes (Fig. 99), but usually not entirely suppressed, even when the 10 milliamperes level is reached at the end of the treatment. This fact is of interest, since current of 10 milliamperes always suppresses respiration at the beginning of the treatment. It appears that when the treatment has been skillfully carried out, the resistance of the patient to the type of stress provided by electric stimulation is increased and a process of adaptation is brought into play, pre-

sumably by the treatment. This tolerance for prolonged electric stimulation is in marked contrast to the behavior of respiration at the beginning of nonconvulsive stimulation, when a current of the level of 10 milliamperes with spikes up to 252 milliamperes invariably suppresses respiration for the first 20 to 40 seconds in all cases.

In lengthening such combined convulsive-nonconvulsive treatments further, it is important to realize the patient's limitations of endurance so as not to push stress beyond the limits of adaptation to the point of exhaustion. Simultaneous observation of respirograph tracings and careful observation of the color and the pulse of the patient are useful guideposts. The advantage of a respirograph tracing is that it reveals disturbance and

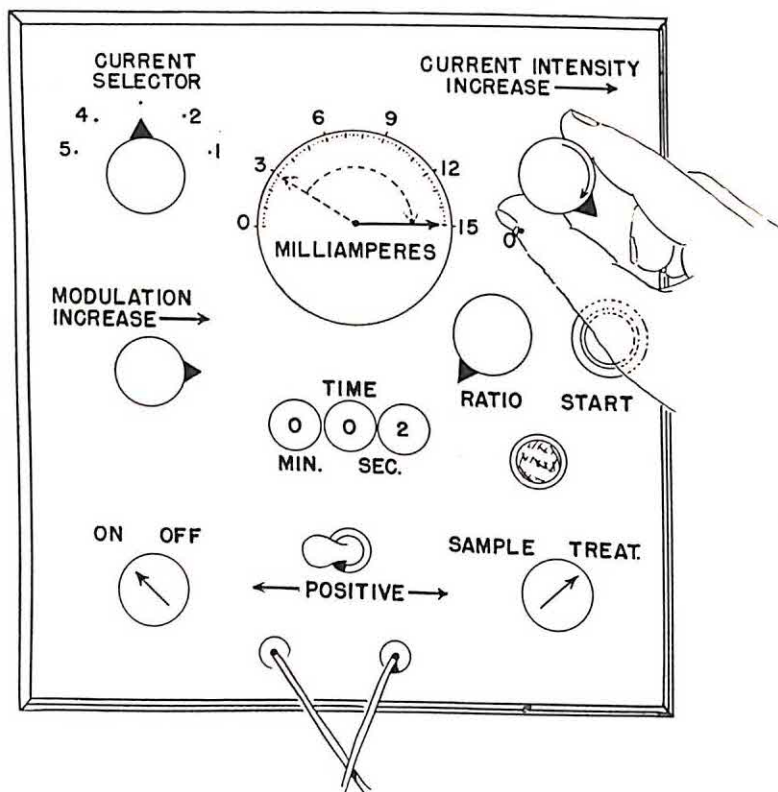


Fig. 117. Electric coma, Step II. Raise current within 2 seconds to 15 MA.

excessive stress before the color of the patient or change in his pulse rate indicates the sequelae of his respiratory stress in the form of cyanosis and anoxia, and therefore provides warning signs before that crucial time.

The treatment described above is the standard treatment I employ in depressions and other mental illnesses complicated by marked epinephrine-precipitable anxiety. It has a marked antidepressant effect as well as a marked anxiety-reducing effect. I also prefer this treatment to standard electroshock in paranoid states, irrespective of whether or not they are complicated by epinephrine-precipitable anxiety.

Combined Convulsive-Nonconvulsive Treatment of Four to Six Minutes' Duration. Four to six-minute treatments, like the preceding three-minute treatment, are given on the Reiter CW 47 machine through fixed bi-

temporoparietal electrodes. The advantage of the extension of the non-convulsive phase of the treatment is the relatively greater relief of anxiety which it affords. It is best to carry out these treatments under respirograph control so as to be sure that sufficient exchange of alveolar air is taking place. Figure 114 shows a treatment of $4\frac{1}{2}$ minutes' duration. It is in all respects similar to the standard three-minute treatment; polarity is reversed every 20 seconds beginning at 1 minute and 40 seconds until the end of the fourth minute. If possible, the current is raised to 5 milliamperes at the end of the second minute. In the case shown in Fig. 114, this proved

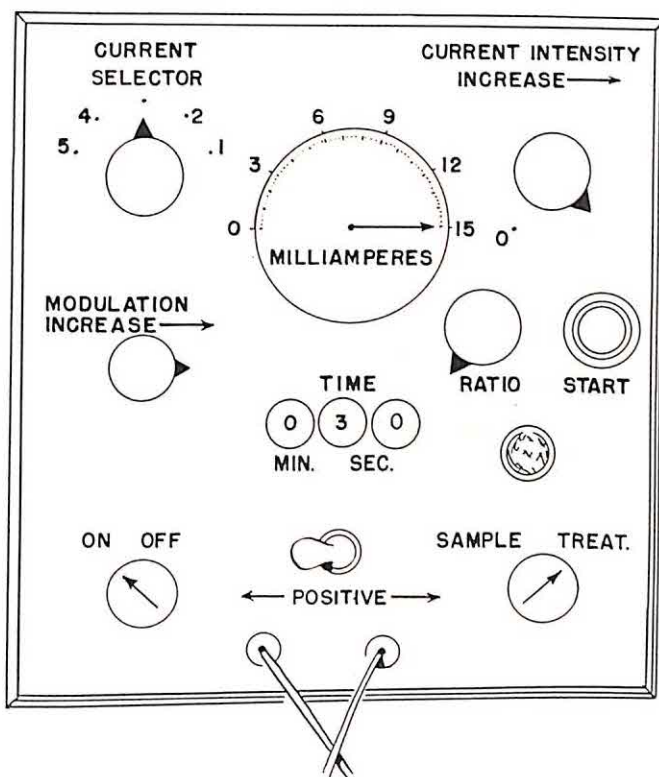


Fig. 118. Electric coma, Step III. Hold current at 15 MA for 30 seconds.

to be too stressful for the expiratory activity, and the current was therefore reduced to $2\frac{1}{2}$ milliamperes 50 seconds later, followed by immediate reexpansion of expiration as soon as the $2\frac{1}{2}$ milliamperes mark was reached. At 3 minutes and 50 seconds, the current was again raised, this time only to 4 milliamperes, but restriction of expirations recurred. Expirations expanded only slightly when the current was lowered to 3 milliamperes, but became normal as soon as the current was turned off $22\frac{1}{2}$ seconds later. It is very interesting to note the perfect correlation between the strength of the current and the degree of restriction of expiration when the degrees of restriction caused by currents of 3, 4, and 5 milliamperes respectively are compared with one another (Fig. 114).

Combined Convulsive-Nonconvulsive Treatment of Seven to Twelve Minutes' Duration ("Electric Coma"). This treatment is given with the Reiter RC 47 machine through manually movable electrodes, which are

operated by an assistant or nurse according to the instructions of the therapist. Moving the electrodes from the bi-temporoparietal placement upward and forward along an S-shaped curve (see Fig. 65) reduces the action of the current, thus providing an additional source of control of the intensity of the stimulation. In addition, this movement of the electrodes presumably assures a wider irradiation of stimulation throughout the brain. During most of the treatment, however, the electrodes should travel about the posterior frontal part of this path. Whenever there is a

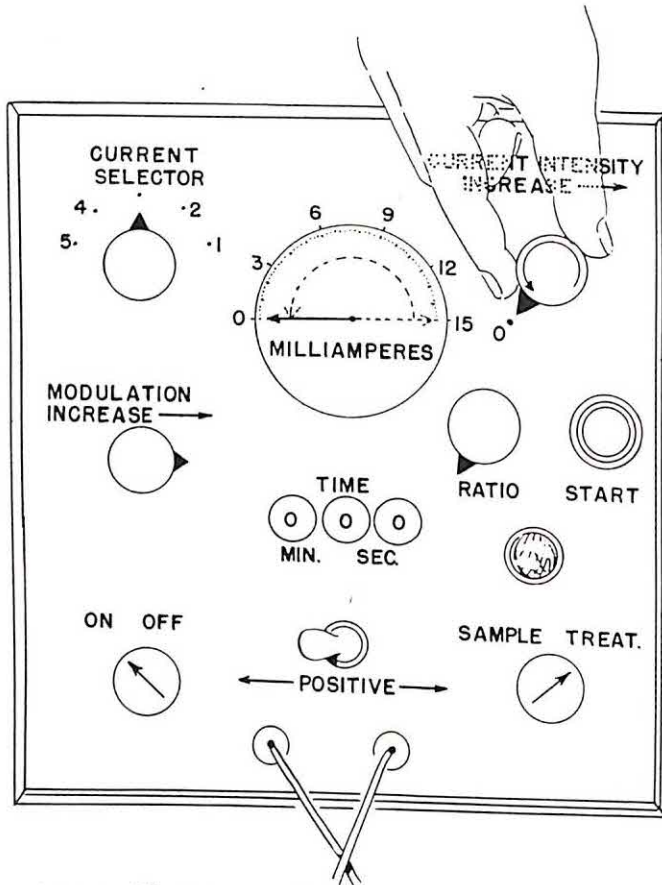


Fig. 119. Electric coma, Step IV. Reduce current to zero.

build-up of clonic bursts heralding the possible advent of a secondary seizure, the electrodes should be moved forward and upward, and whenever the patient becomes too restless and shows signs of awakening, the electrodes should be moved backward and downward to a bi-temporoparietal position.

Before the treatment, the patient should be given 1/100 to 1/50 grain of atropine in order to reduce salivation during treatment. The treatment is initiated by a partly modified tonic-clonic seizure which is best elicited with bi-temporoparietal placement of the electrodes. Clonus is suppressed for 30 seconds by maintaining the stimulating current in that area. Then the current is reduced to zero and the clonus allowed to break through for the remainder of the time span of the convulsion (Fig. 115). At the end

of the seizure, or if the clonus of the jaws is not violent toward the end of the clonic phase, a Guedel airway, which had been used as a tongue protector up to this point, should now be fully inserted. As soon as the airway is in place, the nonconvulsive phase of the treatment can begin with electrodes held in posterior frontal position—at about the level of the coronal suture, midway between the orbit and the ear (at about the middle of the S-shaped line indicated in Fig. 65)—using this area as a pivotal

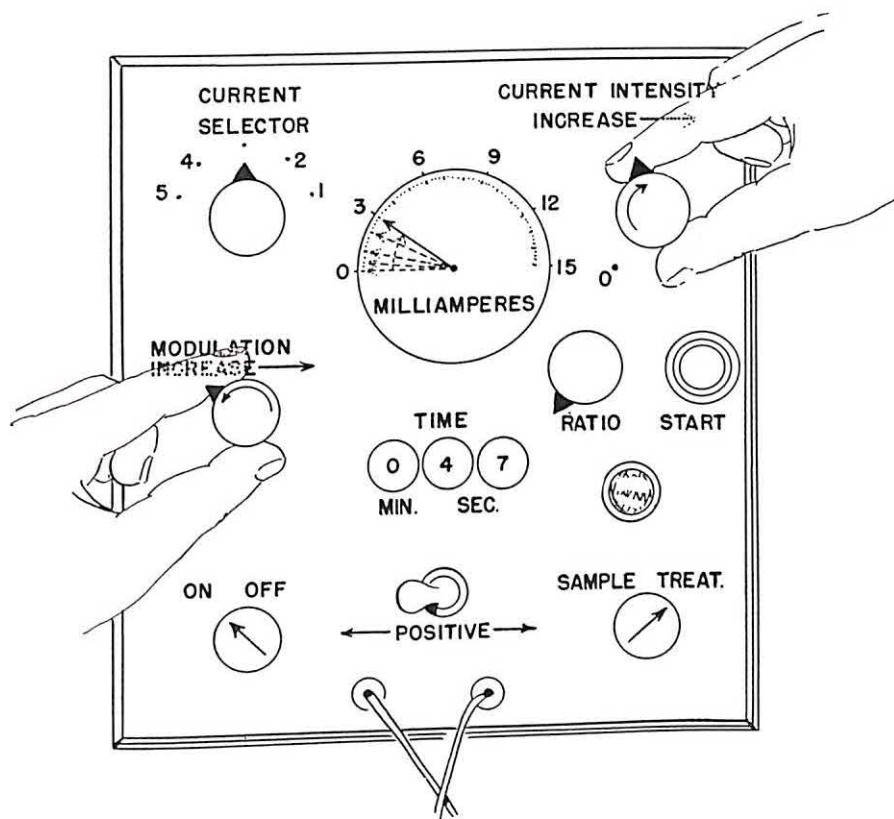


Fig. 120. Electric coma, Step V. After a brief pause necessary for the proper insertion of the airway, turn modulation control dial to slow (counterclockwise to 10 o'clock), and raise current in slow gradual steps, beginning at $\frac{1}{2}$ MA, to 3 MA.

point for occasional movements in accordance with the needs of the treatment as outlined above.

It is advisable to carry out this treatment with respirograph control. A second assistant should watch the tracings and analyze them, giving timely warnings to the therapist who works the control dials of the machine, manipulates the airway, and watches the patient. Needless to say, suction and a tracheal tube should be available, although their use will only rarely be required, especially if the airway is placed far enough backward. The therapist must make certain that the airway is open at all times. If it slips forward or upward only a few millimeters, respiration may become obstructed. It may also become obstructed by mucus (in spite of premedication with atropine). This must be immediately remedied by suction and/or replacement by a fresh airway.

Because of the complexity of this treatment, it is more difficult to standardize it precisely than the foregoing briefer forms of treatment. The tracing illustrated in Fig. 115 is a typical sample.* In view of the length of the treatment uneventful sections have been omitted, these omissions being indicated by a gap in the tracing. This treatment lasted 9 minutes and 57 seconds, including the 15 seconds during which the current was shut off for the purpose of inserting the airway at the end of the convulsive seizure (Fig. 115).

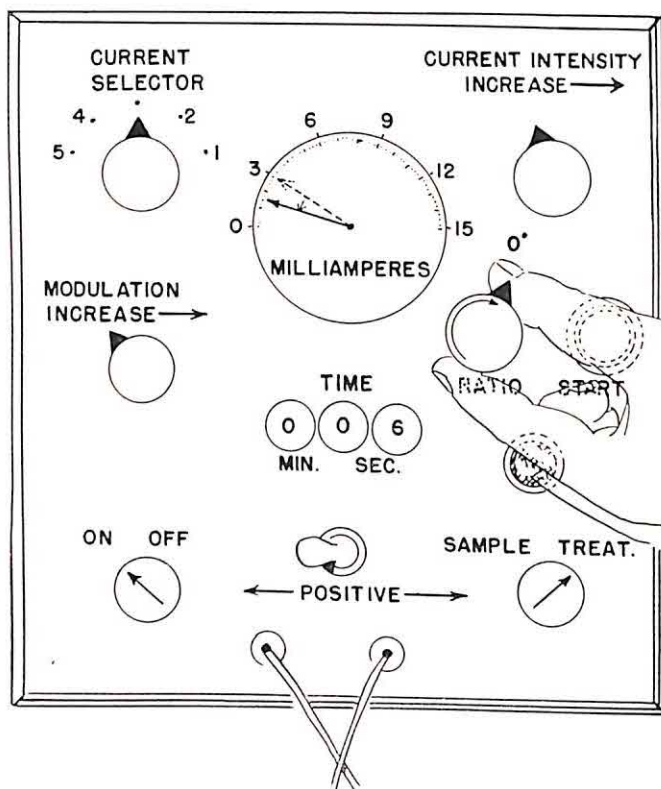


Fig. 121. Electric coma, Step VI. Turn knob marked "ratio" clockwise to 1 o'clock (pointing to the dot marked 0), thus cutting current in half, i.e., to $1\frac{1}{2}$ MA.

The following steps characterize electric coma treatment:

Step I. Set the machine at an average current of 3 milliamperes. Select No. 3 current and continue this type of current throughout the treatment. Begin treatment with fast modulation, modulation knob pointing to 3 o'clock. The ratio dial should be off with the knob pointing to 7 o'clock. Turn on the current (Figs. 115 and 116).

Step II. Raise the current within 2 seconds to an average current of 15 milliamperes (Figs. 115 and 117).

Step III. Maintain the current at a level of 15 milliamperes of average current for 30 seconds (Fig. 118). As long as the current remains at this level, the convulsion will remain tonic.

* Dr. David Landau, who developed this technique of electric coma in collaboration with Dr. William Holt, collaborated in the planning and administration of this sample treatment.

Step IV. Reduce the current to zero by means of the current control dial, leaving the machine on and ready for subsequent further use (Fig. 119). As soon as the current is turned off, clonus will break through for the balance of the time span of the convulsion (Fig. 115). As soon as the jaws are sufficiently limp, insert the airway and make sure that air is exchanged through its core.

Step V. As soon as the airway is inserted, turn the modulation knob to slow (counterclockwise to 10 o'clock), and raise the current gradually over

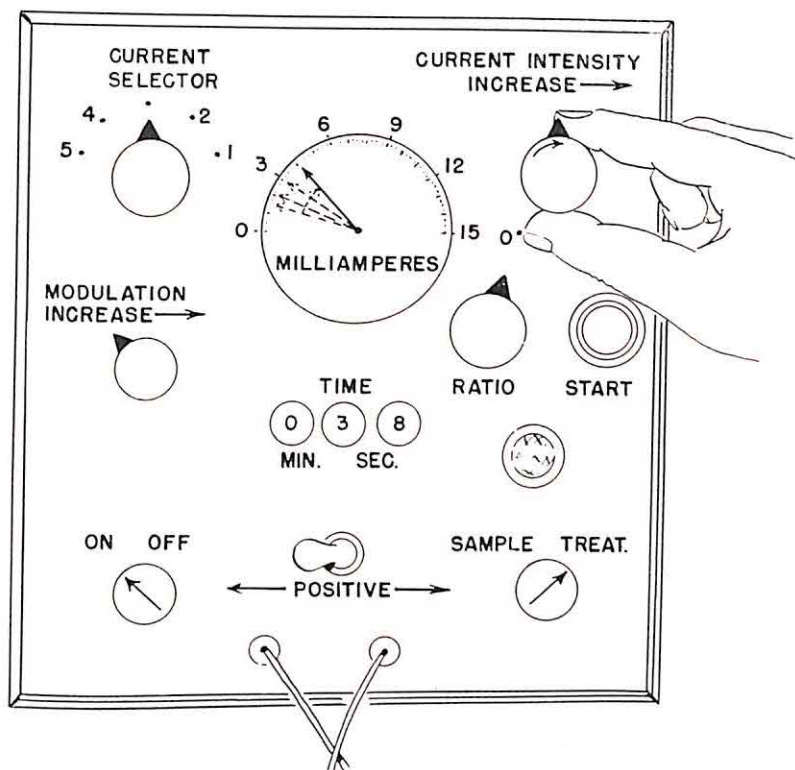


Fig. 122. Electric coma, Step VII. Raise current gradually to 4 MA.

a period of 47 seconds to a level of 3 milliamperes of average current (Fig. 120). Figure 115 indicates the various levels reached during the gradual raising of the current.

Step VI. Turn the ratio knob gradually to the point marked by a circle (clockwise toward 1 o'clock). This will reduce the current to half its value, namely $1\frac{1}{2}$ milliamperes. This operation takes about 6 seconds (Figs. 115 and 121).

Step VII. Raise the current gradually to an average level of 4 milliamperes, leaving the current at that level for as long as coma continues uneventfully with the patient showing neither signs of awakening nor excessively violent myoclonic bursts heralding the possible precipitation of a secondary convulsive seizure (Fig. 122). Minor myoclonic activity will continue throughout most of the electric coma treatment, as is clearly indicated by the spikiness of the kymograph tracing (Fig. 115) compared

with its appearance during the nonconvulsive phase of other forms of combined treatment (compare with Figs. 99, 111, 112, 114).

Step VIII. Increase the speed of the modulation by turning the modulation knob clockwise to 12 o'clock (Fig. 123). This will slightly increase the physiologic effect of the treatment, especially the incidence of myoclonic activity (Fig. 115), but will increase the depth of the coma.

If, however, the patient is showing signs of awakening which not infrequently tends to occur during this phase of the treatment, it is wise to in-

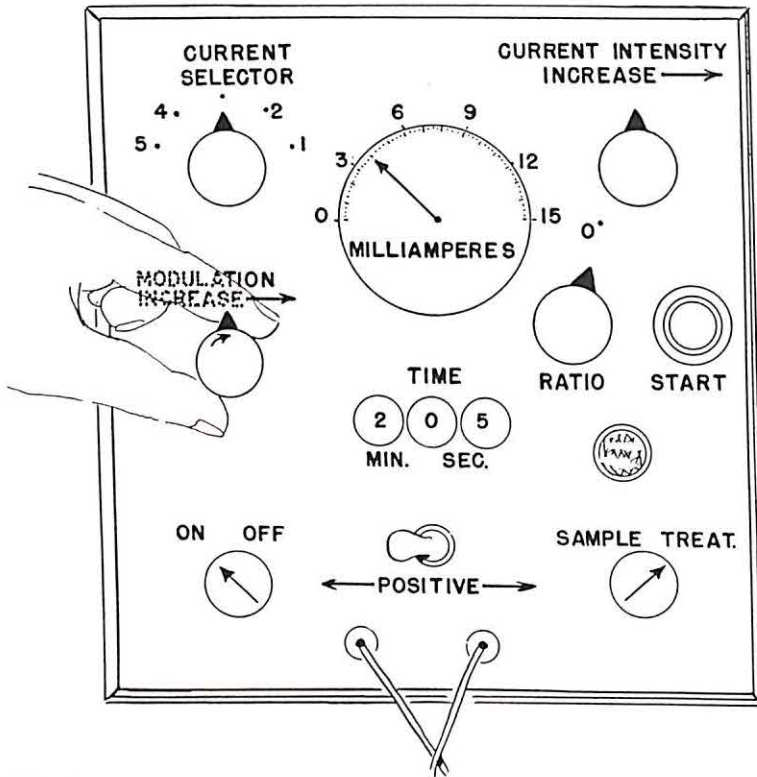


Fig. 123. Electric coma, Step VIII. Increase speed of modulation by turning modulation control dial clockwise to 12 o'clock.

crease the current further, but only for brief periods. This machine contains a particularly helpful device for brief increases of current in the form of a button at the right margin of the machine labeled "start," which temporarily suspends the action of the ratio knob that had been cutting the effective current in half (see Step VI).

Step IX. Increase the current to twice its value by pressing the "start" button and holding it down for 2 seconds at a time (Fig. 124). This will increase the myoclonic activity (Fig. 115) and deepen the comatose state. Sometimes, as in the case of this treatment, one such burst is sufficient to maintain adequate depth of coma for quite a while; in other cases, such bursts must be repeated.

Step X. After one or several bursts of doubling the current, adequate depth of coma can usually be maintained for periods of considerable length by varying the current only slightly between 3 and 4 milliamperes of aver-

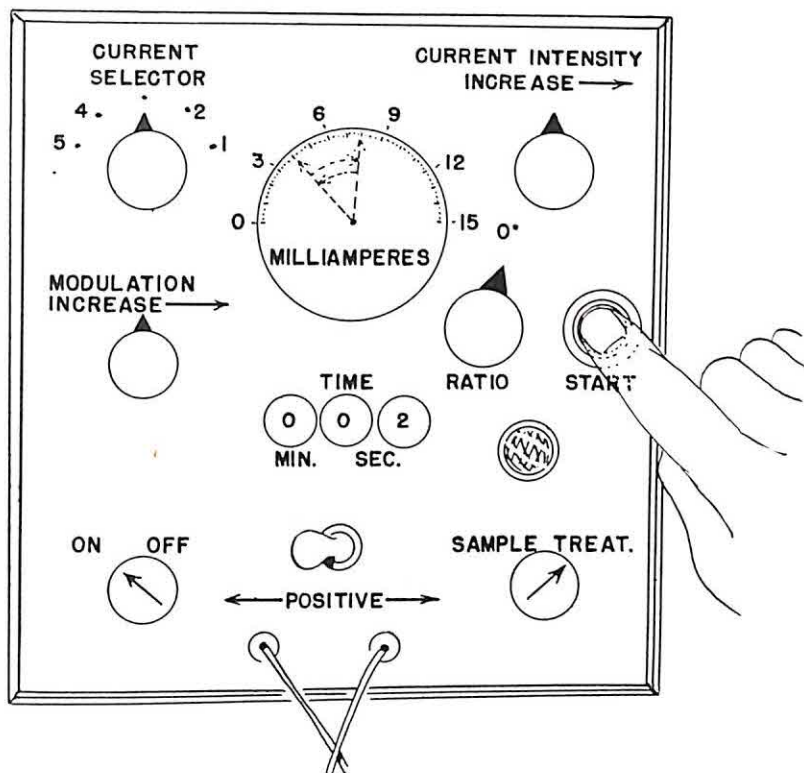


Fig. 124. Electric coma, Step IX. Press button marked "start" for 2 seconds at a time, thus rapidly doubling the current for that period. Repeat this maneuver as often as necessary.

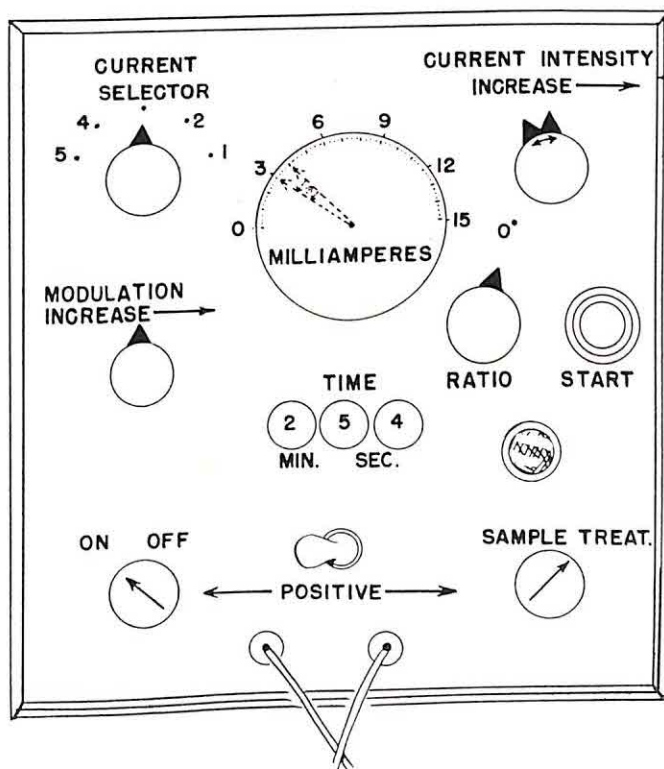


Fig. 125. Electric coma, Step X. Keep treatment current between 3 and 4 MA adapted to the treatment needs of the patient (see text).

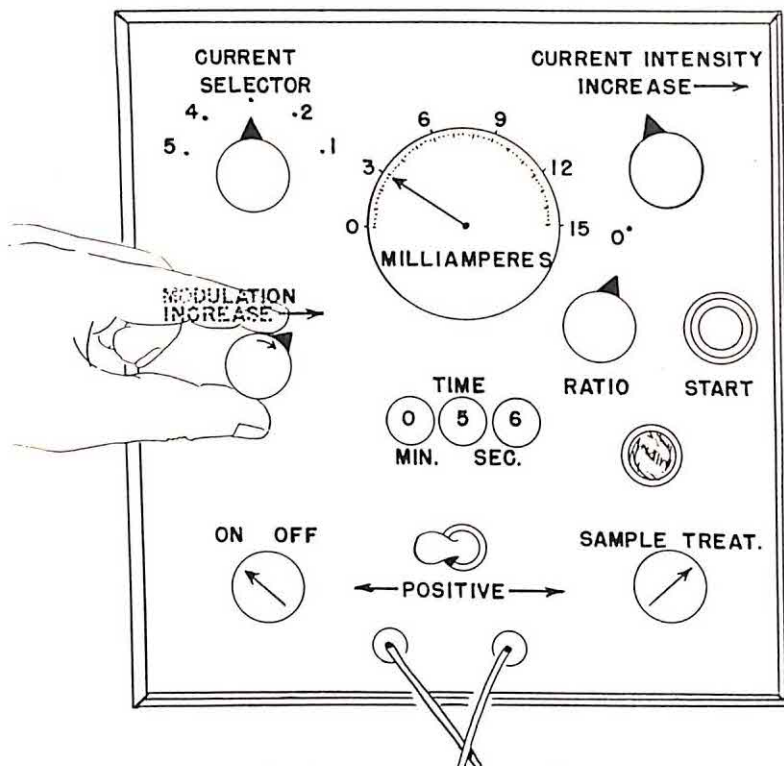


Fig. 126. Electric coma, Step XI. Stabilize current at 3 MA but increase frequency of modulation, by turning modulation control dial clockwise to 2 o'clock.

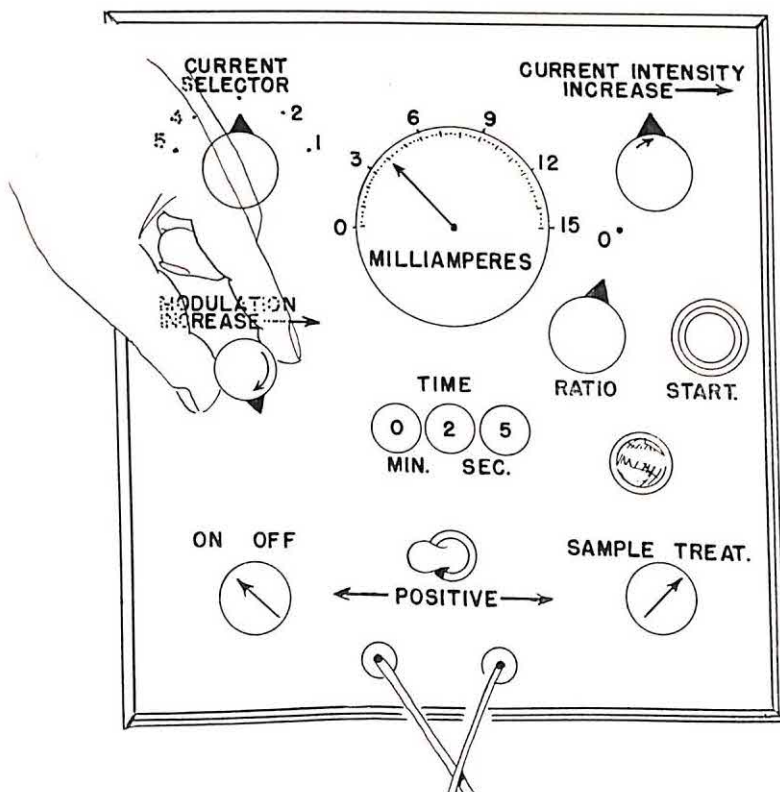


Fig. 127. Electric coma, Step XII. Raise treatment current to 4 MA and increase frequency of modulation to maximal, by turning modulation control dial clockwise to 5 o'clock.

age current (Fig. 125). It was possible to keep adequate treatment going uneventfully at this phase (fifth to eighth minute) without further changes for 2 minutes and 54 seconds in the case illustrated; in other cases, the coma could be maintained for 3 to 4 minutes by maintaining the current within this narrow range of milliamperage. In the present instance, the current was lowered to 3 milliamperes whenever myoclonic spikes increased, and raised to 4 milliamperes whenever the patient showed signs of stirring and waking.

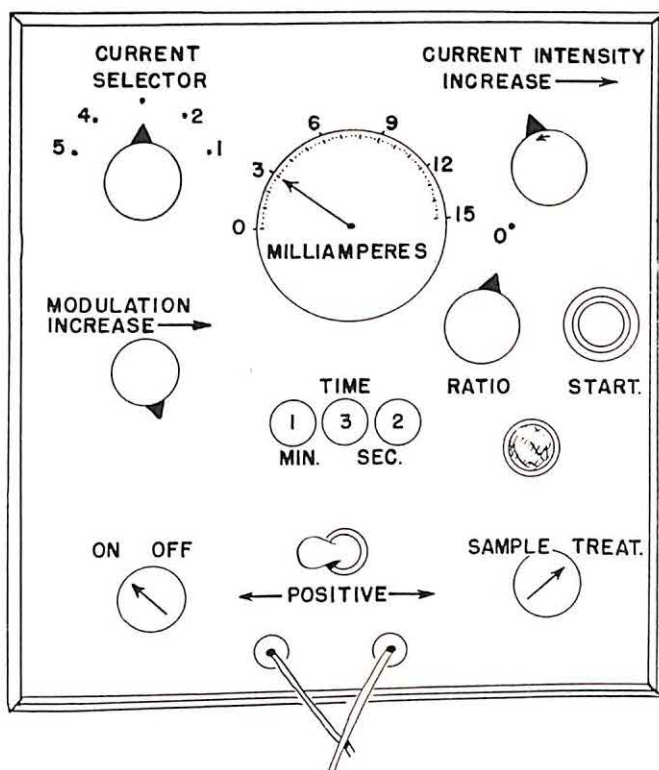


Fig. 128. Electric coma, Step XIII. Decrease current to 3 MA and continue treatment until patient stirs in the process of waking.

Step XI. At this phase of treatment, modulation should be further increased gradually, the first step being a change to about 2 o'clock in a clockwise direction (Fig. 126). This change will sometimes inhibit expiration as in the case illustrated here (Fig. 115), in which, however, expirations returned to their former depth within 15 seconds, thus not necessitating any further action. If expirations should remain reduced for 30 seconds, the modulation should either be slowed down, or the current reduced, or the electrodes moved forward and/or upward. Sometimes all three of these measures must be carried out. It will, however, only rarely be necessary to shut off the current and interrupt the treatment in order to free respiratory activity.

Step XII. Increase current to 4 milliamperes, provided respiration has been adequate, and increase the modulation to the maximally fast range by turning the knob clockwise to 5 o'clock (Fig. 127).

Step XIII. Reduce the current to 3 milliamperes and keep at that level until the patient shows signs of stirring and waking, which in this case took 1 minute and 32 seconds. Then turn the current off.

This is the electric treatment of choice in schizophrenic psychoses either as a less effective but also less cumbersome preliminary alternative to insulin coma treatment or if insulin coma treatment has brought about only slight or moderate improvement. It is sometimes also successful in cases which have relapsed after frontal lobotomy (see Chapters VII and XII).

INSULIN COMA TREATMENT

Insulin coma treatment is by far the most effective treatment of schizophrenia available today. The skill—medical, neurologic, and psychiatric—required for the proper administration of the treatment is generally underrated. This combined skill must enable the physician to administer comas of a maximal depth with a maximal degree of safety for the patient, and then to reinforce the resulting mental and emotional change and make that change meaningful for the patient by intensive psychotherapy. The routine repetition of the same coma dose of insulin that may have been adequate at one phase of treatment may subsequently become either dangerous or ineffectual. If in subsequent phases of treatment this dose turns out to be too high, the patient is exposed to unnecessary danger; if this dose later turns out to be too low, the treatment may remain an ineffectual gesture. I have seen many a paranoid schizophrenic patient who failed to gain further improvement when because of routine repetition of the same dose of insulin he began to achieve only light comas, manifested by mere thalamic and/or posterior hypothalamic release; but whose improvement burgeoned forth at a gratifying rate as soon as the dose of insulin was increased sufficiently to produce comas of maximal depth. If the physician then succeeds in utilizing this improvement and making it meaningful by means of skillful psychotherapy in all its non-verbal as well as verbal aspects, a truly powerful tool in the conquest of schizophrenia is in his hands. Thus the results of insulin coma treatment in the hands of an experienced, if not a master therapist, are far superior to the overall statistical figures given in Chapter XII, impressive though they are.

Insulin coma treatments are administered once daily for six days during each week, every seventh day being a day of rest. A full series of insulin coma treatments in schizophrenic patients should comprise 60 deep comas, unless full recovery supervenes earlier. The series of insulin comas should be preceded by a series of 6 to 20 subcomas, administered once daily over a period of about one to three weeks. During this preliminary subcoma period the subcomas are gradually deepened by a slight daily increase of the dose until the coma dose is reached, which varies considerably from patient to patient. It is frequently desirable, especially in patients who have not fully recovered by the end of the series of deep comas, to add another phase of 6 to 14 subcomas administered once daily at the end of the treatment series. During this final phase the daily dose of insulin should be gradually reduced, somewhat faster than it was built up during the initial subcoma period.

The full treatment therefore usually requires about 75 to 110 treatment days, extending over a period of about 3 or 4 months. Patients

receiving insulin coma therapy must be hospitalized; a period of 3 or 4 months' residence on an insulin ward or equivalent supervised accommodation must therefore be planned for.

The first dose of insulin injected intramuscularly in any patient on the first treatment day should never exceed 10 units (see Chapter X); a fatality has been reported from an initial dose as low as 50 units. In general I regard any initial dose larger than 10 units as dangerous and unwarranted. On subsequent days, the daily dose of insulin should be raised gradually, at first by not more than 10 units a day; later, especially when reactions are well tolerated, by 20 units per day until coma levels are reached. An experienced therapist may at times regard it as safe to increase the daily dose by 30 units per day, especially in insulin resistant patients.

During the subcoma phase of the series, interruption by oral administration of glucose in orange juice should be carried out at first after three hours, later during the fourth hour, finally at the end of the fourth hour after injection of insulin. Approximately 1 gm. of sugar must be given for every unit of insulin that has been injected.

As the daily dose of insulin gradually increases, coma finally supervenes, at first usually at the end of the third or during the fourth hour after injection. Coma will generally supervene at doses between 100 and 400 units of insulin. Once an adequate coma dose has been reached, that same dose may again be given on subsequent treatment days without requiring much modification; however, the physician must at all times be alert in continually reevaluating the dose and its effects in terms of the treatment needs and the safety requirements of his patient. Thorough familiarity with the neurophysiologic aspects of insulin coma as described in the insulin section of Chapter IX is essential for the proper management of the treatment. If at any time during the course of the treatment series the progress of coma should become too precipitous, as recognizable on the basis of the proper evaluation of the neurologic manifestations (see Chapter IX), and/or if termination of coma should at any time become difficult or delayed, the next day's dose of insulin must be reduced. On the other hand, if progress into coma after the day's injection of insulin should become too slow, or if the coma should fail to reach adequate depth within the safe maximum of one hour allotted to the comatose state, the next dose of insulin must be raised appropriately for the sake of giving the patient maximal therapeutic benefit. As stated above, this applies particularly to paranoid schizophrenics; anxious and confused patients, especially at the beginning of the coma series, sometimes do better with relatively lighter comas. The physician must therefore continuously evaluate not only the quality and degree of the comas administered, but also their psychophysiologic effect upon the patient as treatment progresses. There are patients who continue to achieve adequate and therapeutically effective comas with the same dose of insulin throughout the entire treatment series; others become increasingly sensitized to the dose, so that during the second half of the series the dosage of insulin required for adequate depth of coma may be half of what was required for the same effect at the beginning of the series. Others may become somewhat resistant to insulin, requiring increasing doses, usually at about the end of the first half of the series.

It is very important not to allow the patient to remain in coma for too

long a period at the beginning of the treatment series, since fatalities have frequently resulted from permitting comas as long as one hour in duration to develop early in the treatment series, although this constitutes a perfectly safe duration later in the series when therapy has gradually progressed to this stage. The first coma must never be permitted to exceed 15 minutes. If the patient is aroused promptly and with ease from the first coma by intravenous injection of 12.5 gm. of glucose, the second coma, to be administered the following day, may be allowed to last 30 minutes; the third coma—on the third day—45 minutes; and—again provided that resuscitation has been prompt after each of these preliminary periods—the fourth coma, on the fourth day of the coma series, is the first one which should be allowed to extend for the full hour, provided that the medullary phase has at no time lasted longer than 15 minutes (see Chapter IX). From then on comas should be allowed to last one full hour each, including a maximum of 15 minutes for the deepest (medullary) phase.

In some patients coma may be very difficult to establish. The reasons for such "insulin resistance" are not as yet clearly understood, involving a good many unknown factors (Harris¹⁵¹). However, the safest method of overcoming insulin resistance is to allow the period of hypoglycemia to lengthen gradually into the fifth hour. It must be thoroughly appreciated, however, that fifth hour comas are dangerous, and it is therefore necessary to consider that time spent in coma during the fifth hour after injection is equivalent proportionally to twice the duration of a normal third or fourth hour coma. Thus, half an hour spent in coma during the fifth hour after injection is equivalent to one full hour spent in coma during the third or fourth hour after injection. Correspondingly, the maximum safety limit for the deepest (medullary) phase of coma is only $7\frac{1}{2}$ minutes during the fifth hour, while it is 15 minutes during the third or fourth hour after injection of insulin.

Another method of overcoming insulin resistance is known as "swinging of the dose," i.e., the present dose is first reduced by one-half; then on the following day the dose is increased to three fourths of the original dose which had failed to bring about coma; on the third day the original high dose is again employed. Other aids are the use of sedation on the preceding night (sodium amytal, 3 grains before bedtime), or the administration of ammonium chloride, 0.5 gm. (enteric-coated tablets) four times daily orally.

Interruption of the coma should be carried out preferably by intravenously administered glucose, 12.5 to 25 gm. (I prefer to give 25 gm.), to be followed by oral administration of the additional amount of glucose required—a total of 1 gm. of glucose for every unit of insulin administered. This method has certain advantages over slow interruption by gavage (Cameron and Hoskins¹⁴⁸). Intravenous interruption clears the patient more promptly and completely, preventing the anxiety, confusion, and depression characteristic of a lingering mild hypoglycemia, and thus leaves him in a more animated state for the remainder of the day; hence, he will eat better and, as a combined result of all these advantages, be less subject to secondary comas. If the patient cannot be aroused within 5 minutes after injection, the intravenous dosage of dextrose should be repeated. If the patient is still comatose within 5 minutes after the second injection, the diagnosis of irreversible coma must be made and the entire regimen

for the termination of irreversible coma must swing into action (Chapter X).

The physician carrying out insulin coma therapy must be thoroughly familiar with all the physiologic phenomena involved. These have been described at length in Chapter IX (p. 122) and therefore will not be repeated, except for the emphatic statement that all facts put forth in Chapter IX are essential to the proper administration of the practical technique of insulin coma treatment. The same may be said about the technique of interruption which is described in Chapter IX, and the safeguards and life-saving measures described in Chapter X. I shall merely reiterate the point here that interruption must be started 10 to 15 minutes earlier if interruption by gavage is used. This is to allow for the longer time of absorption of glucose when this route is employed. If the patient does not awaken by 15 to 20 minutes after attempted interruption by gastric gavage, intravenous interruption is in order; if this proves insufficient to arouse the patient within 5 minutes, the full regimen for resuscitation from irreversible coma must be applied (see Chapter X).

It should be remembered that pulse and blood pressure increase during phases of sympathetic release, and decrease during phases of parasympathetic release (Himwich,¹⁵² Reiner and Scheffen¹⁵⁶). Decline of blood pressure and pulse during maximally deep phases of coma is a danger sign that calls for immediate interruption by vein. A convulsive seizure during any phase of coma always calls for immediate interruption by the intravenous route.

Whenever interruption has been difficult because of slow response of the patient to an adequate dose of glucose, the next day's dose should be reduced by at least 15 per cent, and the progress of coma observed very carefully, so that interruption can be carried out promptly as soon as signs of excessive depth of coma appear or when progress of depth of coma becomes precipitous.

After resuscitation from therapeutic coma, the patient should be observed carefully throughout the remainder of the day and the subsequent night. Secondary comas should be prevented as much as possible, first, by administration of adequate amounts of glucose at termination of the original coma; second, by prompt diagnosis and treatment of secondary hypoglycemic reactions as soon as they appear, and before they deepen into secondary coma. This is particularly important at night, when secondary comas may be easily overlooked unless the patients are awakened every three hours, at which time they should be given small amounts of glucose (100 gm. in orange juice) by mouth, to be increased whenever the patients are found to be perspiring or unusually difficult to arouse.

INSULIN SUBCOMA TREATMENT

In insulin subcoma treatment the patient is subjected to a slight to moderate state of hypoglycemia extending over a three-hour period. This state of hypoglycemia should be allowed to deepen to the point of sopor, but should stop short of coma. Treatments should be given once daily, with the exception of one day of rest per week. The series of treatments should be begun with a dose of 10 units, to be increased by 10 units daily until the initial symptoms of subcoma, usually perspiration, tremors, and sympathetic release, manifest themselves within one half-hour after injection

and are consistently followed by a satisfactory level of subcoma gradually progressing to a slight soporous phase by the time three hours have elapsed. Until sopor short of coma supervenes, there is usually a slight activation of symptoms, first of anxiety and then of depression. In most patients the daily dose need not be increased above 50 units, but in some cases it may have to be increased up to 100 units.

The technique of interruption is simple. If sopor is fairly marked, glucose in orange juice should be given by mouth, 1 gm. of sugar for every unit of insulin injected. If the patient can be readily aroused without administration of 50 per cent glucose, it is best to let a large breakfast rich in carbohydrate do the interrupting. A glass of only slightly sweetened orange juice followed by a heaping bowl of cereal with ample amounts of cream and marmalade, coffee, two eggs, bacon (or cheese), bread and toast with butter, jam or marmalade (*ad lib.*) will be welcome as well as effective. After the breakfast there should be bed rest for two hours. Three and a half hours after interruption the patient should have a light lunch and engage in graded exercise throughout the afternoon. Dinner should be served at 5 P.M. The patients will usually enjoy eating another snack before bedtime at which time they should be allowed ample amounts of food of their own selection.

In acute anxiety states two weeks of treatment are usually sufficient to bring about significant improvement or recovery, especially if the psychotherapeutic rapport established during the treatment phase has been effectively utilized. Patients who have lost a good deal of weight will usually gain weight rapidly, sometimes at the rate of one pound per day. If thirty treatments have not resulted in significant improvement or recovery, treatment should be changed to coma provided such treatments are otherwise indicated.

COMBINED INSULIN COMA—CONVULSIVE ELECTROSHOCK THERAPY

Convulsive electroshock treatment can be combined with insulin coma treatment whenever it is indicated on the basis of the clinical findings, such as a marked depressive component of the illness or prevalence of severe regressive catatonic manifestations, especially when these appear to become released or exacerbated, as they sometimes are in certain cases, particularly at the beginning of the insulin coma series. In most such cases requiring combined insulin coma—electroshock treatment, one convulsive electroshock administered after every third to fifth coma suffices. I prefer to use maximal amounts of alternating current administered through bifrontal leads (400 to 500 milliamperes of average current flowing for 0.6 to 1.0 second) for this purpose. The convulsive electroshock is best administered at the end of the hour of coma, immediately preceding interruption, preferably by intravenous administration of dextrose.

ETHER DRIP INFUSION TREATMENT

This treatment is administered by means of a sterile solution of 1000 cc. of 5 per cent dextrose in normal saline that has been chilled overnight and to which immediately before treatment 25 cc. of ether is added and thoroughly mixed. This mixture is administered by intravenous infusion. A flow of an average of 100 drops per minute should be maintained throughout most of the treatment. It is important not to give the infusion too fast

at the beginning; only 30 drops per minute should be given at the outset. Later, as the patient proves to tolerate the treatment well, the infusion may be allowed to accelerate to 180 drops per minute. Each treatment therefore may take from $2\frac{1}{2}$ to 4 hours. It is very important not to allow the last 50 cc. of the infusion fluid to enter the vein, because in spite of frequent shaking of the mixture, which must be done throughout the treatment, some excess of ether is bound to accumulate at the top of the column of infusion fluid, especially as the infusion fluid warms gradually in the course of its exposure to room temperature. The last 50 cc. in the infusion bottle are usually composed of this highly concentrated residue. Allowing such excessively high concentration of ether to enter the vein may cause local thrombosis or, worse, fatal pulmonary fat embolism (see Chapter X). Treatment should be given daily allowing one or two rest days at the week end. An effective course of treatment may comprise from ten to thirty treatments.

FRONTAL LOBOTOMY AND UNDERCUTTING

This chapter does not purport to be a treatise on the technique of frontal lobotomy since, with all due respect for the pioneer work carried out in this field by non-neurosurgeons, lobotomy should now be regarded as a specialized neurosurgical procedure. However, the neurosurgeon will expect the neuropsychiatrist or the medical man who recommends the operation to establish the indication for the operation, and he will look for guidance from him in regard to the extent of the operation recommended. The neuropsychiatrist and the medical man should therefore have information as to the techniques and types of operation available.

Freeman and Watts¹⁶⁴ carried out four types of frontal lobotomy distinguished by location, extent, and approach to the operation.

1. *The standard operation.* This consists in sectioning almost the entire white matter of the frontal lobes at the level of a plane drawn from the coronal suture to the sphenoid ridge (Fig. 129), through a lateral small opening in the skull not permitting visual control of the operation. Freeman and Watts substituted for visual control an intricate system of guiding landmarks which, though not precise (Mettler et al.¹⁷²), nevertheless permitted a fairly consistent extent of the operation. Freeman and Watts¹⁶⁴ were greatly aided in the relative precision of their sections by their method of introducing contrast medium into the level of section, thus permitting x-ray verification of the plane of section (Figs. 129 and 130). They found the standard operation sufficient for most cases of chronic, particularly involutional, depression, as well as for some cases of schizophrenia, but found this method unnecessarily extensive if applied to patients with intractable pain.

2. *The radical operation* was the second type devised by Freeman and Watts. This consisted in a section made 10 to 15 mm. posterior to the coronal suture and the sphenoid ridge. Figure 130 shows an x-ray of the same patient shown in Fig. 129, who had to be reoperated on because of insufficient relief afforded by the first (standard) operation. Freeman and Watts devised the radical operation especially for severe cases of schizophrenia which had failed to be relieved by the standard operation.

3. *The minimal operation.* This is carried out in the same plane as the standard operation. However, the cut is made downward only, thus limit-

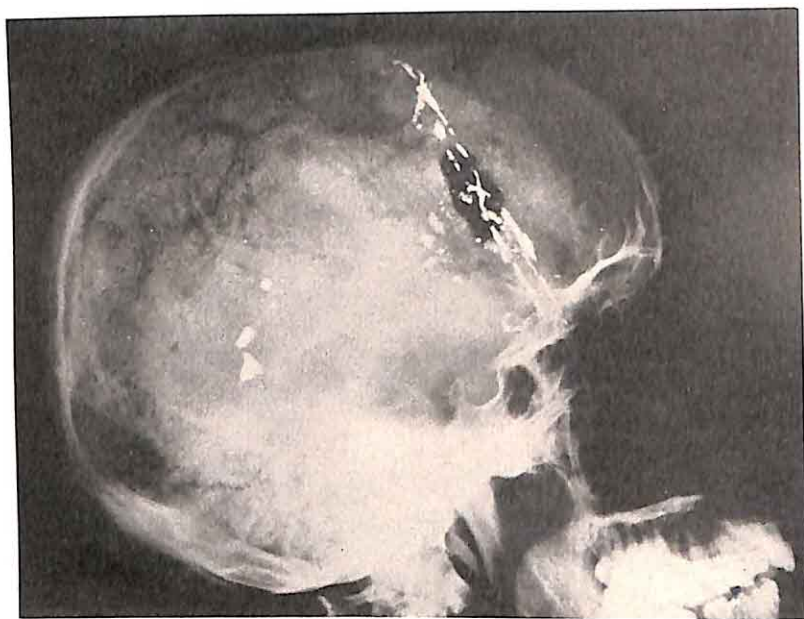


Fig. 129. Lateral roentgenogram showing standard lobotomy. (From Freeman and Watts: *Psychosurgery*, Charles C Thomas.)

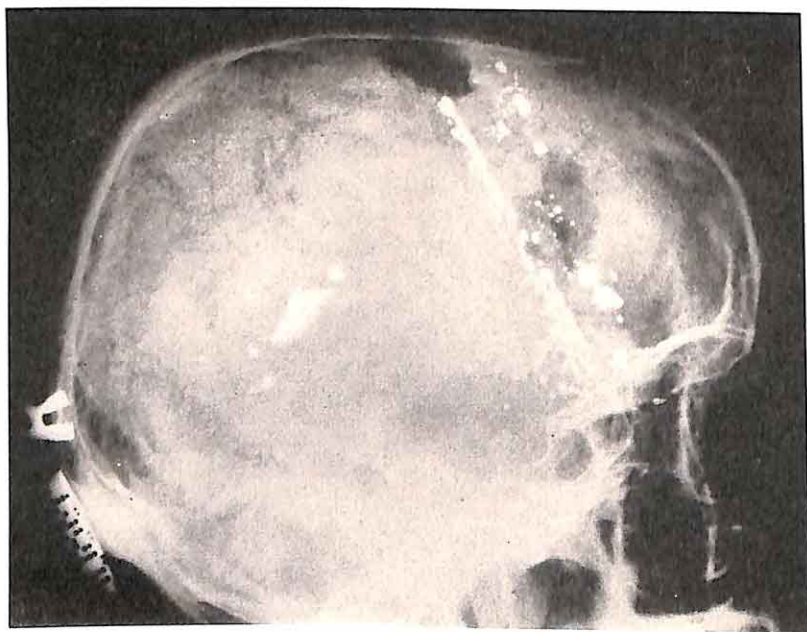


Fig. 130. Lateral roentgenogram of secondary (radical) lobotomy, carried out 10 to 12 mm. posterior to coronal suture and sphenoid ridge. (From Freeman and Watts: *Psychosurgery*, Charles C Thomas.)

ing the sectioning of the frontal white matter to the orbital quadrants. This operation is usually sufficient in cases of intractable pain (Freeman and Watts,¹⁶⁴ page 81).

4. *The transorbital operation.* This operation was first introduced by Fiamberti,^{161, 162} later modified by Freeman.¹⁶³ It is done during the brief period of unconsciousness that follows a convulsive electric shock without further anesthesia, by means of a pointed leukotome entered through the upper conjunctival sac. This tool is tapped lightly through the roof of the orbit into the anterior intracranial fossa to a depth of 5 cm., turned laterally as far as the rim of the orbit will permit, then returned halfway to

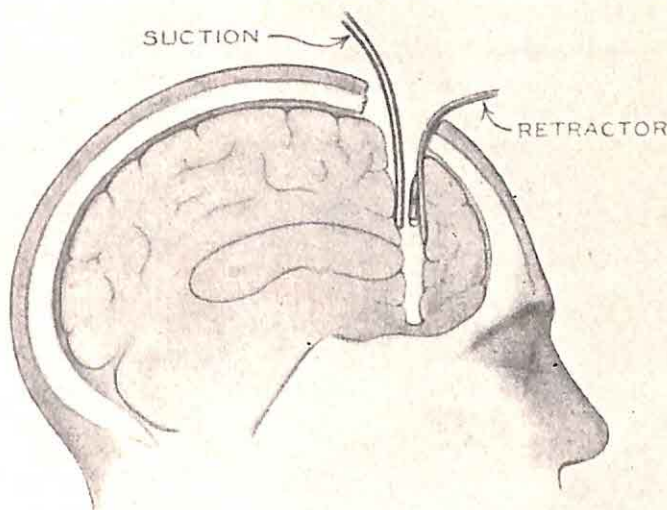


Fig. 131. Poppen's modification of Lyster's open method of frontal lobotomy. The plane of leukotomy is shown as executed by the electrosurgical suction apparatus under direct vision by means of a lighted brain retractor. (From Poppen: *J. Neurosurgery*, vol. 5, 1948.)

its previous position and driven further to a depth of 7 cm., where it is turned 15 to 20 degrees mesially, then turned 30 degrees laterally as well. The handle of the instrument is then elevated so as to be approximately parallel to the floor of the anterior fossa and the turning maneuver repeated over an arc of 30 degrees in mesial direction in order to cut the orbital fibers. The instrument is then removed and prolonged pressure applied to the eyeball to prevent bleeding into the orbit. According to Freeman¹⁶³ the advantages of this method are: no postoperative downgrading, no drastic sequelae, and the fact that it can be performed in a matter of minutes. Freeman recommends the use of transorbital lobotomy in early cases of schizophrenia after shock treatment has failed and in place of maintenance electric shock treatment. If this procedure fails, then more radical surgery may be indicated.

In all these four types of operation the intracerebral sections are carried out without visual control, and hence specific dissection of the convolutional ramifications of the tracts of white matter involved cannot be carried out. This, however, is indicated particularly in regard to the uncinate

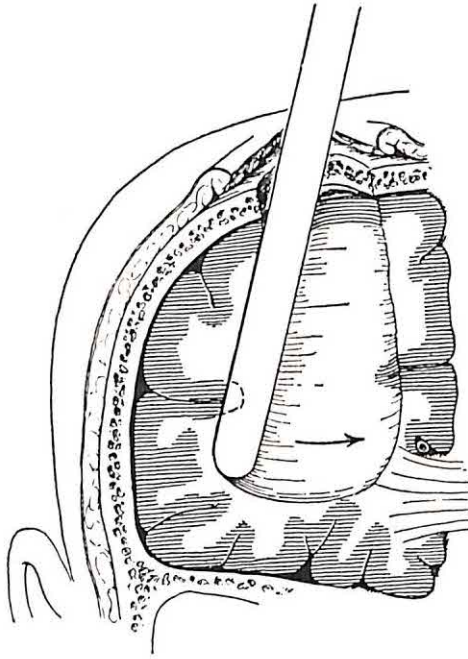


Fig. 132. Poppen's modification. The extent of the first incision with spatula is shown. (From Poppen: *J. Neurosurgery*, vol. 5, 1948.)

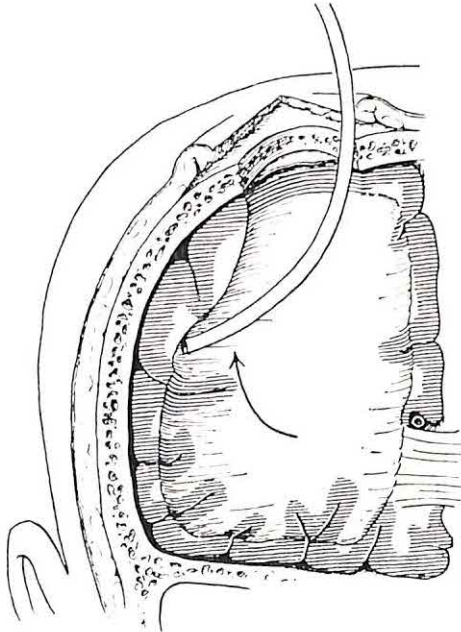


Fig. 133. Poppen's modification. Subsequent dissection of folia of convolutional white matter with a curved suction tip. (From Poppen: *J. Neurosurgery*, vol. 5, 1948.)

tract which dips deeply into the convolutional leaves of the white matter (see Fig. 44, Chapter IX). Another disadvantage is that hemorrhage cannot be immediately recognized and controlled. These two requirements are filled by the open operation devised by Lysterly¹⁷¹ and Poppen¹⁷⁷ (Fig. 131). Poppen dissects the bulk of the central white matter with a straight instrument (Fig. 132) and then dissects the individual folia of white matter with a curved instrument (Fig. 133); thus the extent of the operation can be controlled by direct visual means.

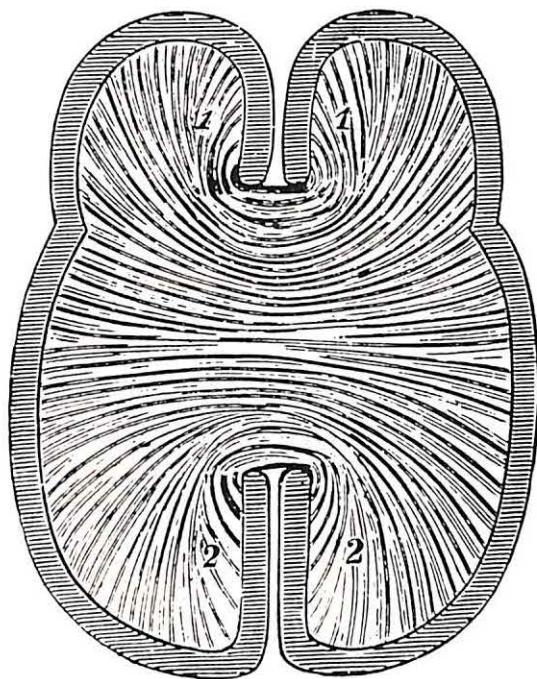


Fig. 134. Distribution of the commissural fibers of the corpus callosum. 1, Frontal ramifications. 2, Occipital ramifications. The neurosurgeon should take pains to destroy as few of the frontal callosal commissural fibers as possible. (From Kopsch: *Rauber's Lehrbuch der Anatomie des Menschen*, vol. 5.)

Modifications of Frontal Lobotomy. It has gradually become recognized that sectioning of the superior longitudinal fasciculus, as well as of the rich supracallosal association and the callosal commissural systems (Fig. 134), is not only unnecessary for achievement of a good therapeutic result but largely responsible for the undesirable side effects following the operation, particularly apathy and inertia. Operations were therefore devised which prevented unnecessary damage to these association and commissural systems. Scoville's¹⁸¹ method of undercutting of specific areas, particularly at the medial, orbital, and anterior surface of the frontal lobes, was the first practical step in this direction, since the method of topectomy which was devised for the same purpose proved impractical in view of the increased incidence of seizures following cortical operations. Ward¹⁸⁹ and Livingston¹⁷⁰ have established the fact that undercutting of as little as the anterior cingulate gyrus (area 24) only may have considerable beneficial effect. However, undercutting operations alone are frequently insufficient,

especially in the severe forms of illnesses in which psychosurgery is indicated. Hannibal Hamlin¹⁶⁷ devised a combined operation in which he attempted through an open Lyster-Poppen approach to aim for the specific important projection and association systems while doing as little damage as possible to the supracallosal association and the callosal commissural systems. In addition to an undercutting procedure as devised by Scoville, the operation is carried out in four steps (Fig. 135). Two of these steps may be sufficient in pain problems or some chronic depressions; all four

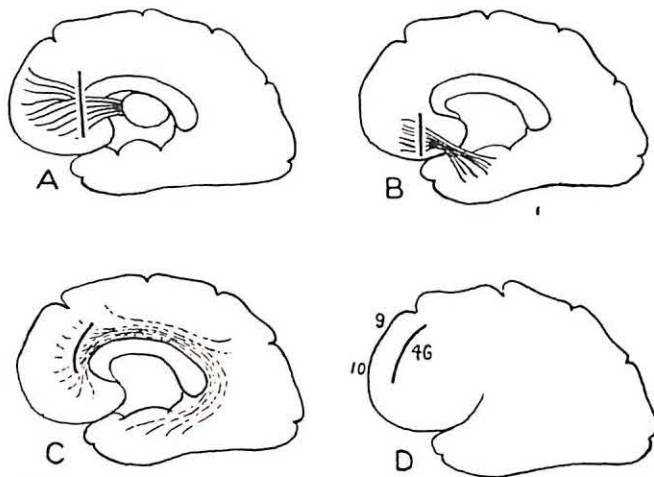


Fig. 135. The four essential and decisive component steps of frontal lobotomy:

A. Transection of thalamofrontal tract.

B. Transection of uncinate tract.

C. Transection of cingulum.

D. Undercutting of areas 9 and 10.

Two of these steps may be sufficient but all four are required in the most severe group of cases. (From H. Hamlin.)

are necessary in schizophrenia and in most chronic obsessive-compulsive states. These steps are:

1. Cutting of the thalamofrontal tract (Fig. 135A).
2. Cutting of the uncinate tract with as careful dissection of the folia of white matter as possible (Fig. 135B).
3. Section of the cingulum at the mesial margin of the central white matter of the frontal lobe (Fig. 135C).
4. Undercutting of areas 9 and 10 (Fig. 135D).

More recently, Bockoven, Levine, Greenblatt, Solomon, Poppen and their coworkers^{160, 169, 178} found that a bimodal operation—consisting in sectioning of the medial white matter only, including its upper and lower parts, thus sectioning the cingulum fairly completely as well as the medial part of the uncinate tract, but only the lower medial extension of the thalamofrontal tract—brought about good therapeutic results with only minimal side effects. For severe types of cases, however, Poppen¹⁷⁸ rightly still takes recourse to his complete standard operation. Grantham¹⁶⁵ established the fact that, for the relief of pain, bimodal operations, including some that were limited to only the lower medial quadrant of each hemisphere, were sufficient.

In performing any of the less extensive procedures, it is wise to explain to the family that this procedure is carried out in order to make sure that excessive depletion of the patient will be avoided but that the first operation therefore may only be partially successful, thus making sure in advance that the family will give permission for a second operation if the less extensive first operation should fail. If the family is unlikely to give permission for a second operation, it may be wise not to subject the family to the frustration of a poor result but rather to gauge the operation as accurately as possible in regard to the extent that will be required to relieve the condition. If warned beforehand, however, families practically always give permission for a second operation.

Van Wagenen, Bailey, Small, Ingraham, and C. T. Liu^{159, 188} and Putnam¹⁷⁹ have developed a method of pretesting the prospective response to lobotomy by injecting a 2 per cent solution of procaine or 10 per cent procaine base in iodized oil into the frontal white matter by the transorbital route. Putnam's method utilizing iodized oil allows also x-ray verification of the extent of the procaine infiltration.

The most promising development in the direction of perfecting a method of fractional lobotomy adapted to the requirements of the individual patient is Grantham's¹⁶⁵ method of focal electrocoagulation. He uses high frequency electric currents applied through a fine needle insulated everywhere except at the tip. With this method he was able to approach and destroy a desired area without damage to other areas of the hemisphere, especially to the cortex. He uses pneumoencephalograms taken during the operation (using only 10 cc. of air) as guides for the exact placement of each lesion. If necessary, more such lesions can be added in two or three stages if subsequent study of the patient indicates that a more extensive lobotomy is required.

SURGERY OF THE TEMPORAL LOBE

Amputation of the anterior parts of both temporal lobes (temporal lobectomy) was introduced by Bailey and Gibbs¹⁵⁸ for the relief of psychomotor epilepsy. Temporal lobotomy was explored by Obrador¹⁷⁶ in schizophrenics, with inconclusive results. Thompson and Walker¹⁸⁷ reported increased placidity in some disturbed schizophrenic patients after extirpation of the amygdaloid complex of the temporal lobes. Williams and Freeman¹⁹⁰ reported relief of persistent hallucinatory experiences by removal of the amygdaloid nucleus. Recently Scoville, Dunsmore, Liberson, Henry, and Pepe¹⁸² studied the effect of combined medial temporal lobotomy and uncotomy in the treatment of psychotic states. By and large it may be stated that the problem of temporal lobectomy, lobotomy, uncotomy, and surgery of the amygdaloid nucleus is still in the experimental developmental stage both as regards the relief of psychomotor epilepsy and in regard to relieving patients suffering from major psychoses, especially schizophrenia. Indications, therefore, cannot yet precisely be determined. However, I would consider it advisable to undertake unilateral or bilateral temporal lobectomy in cases of psychomotor epilepsy with proven anterior temporal lobe foci, as demonstrated by the typical spikes, particularly in the sleep record, and to investigate the effects of extirpation of the amygdaloid complex in cases with persistent and disabling hallucinatory experiences in schizophrenic and paranoid states.

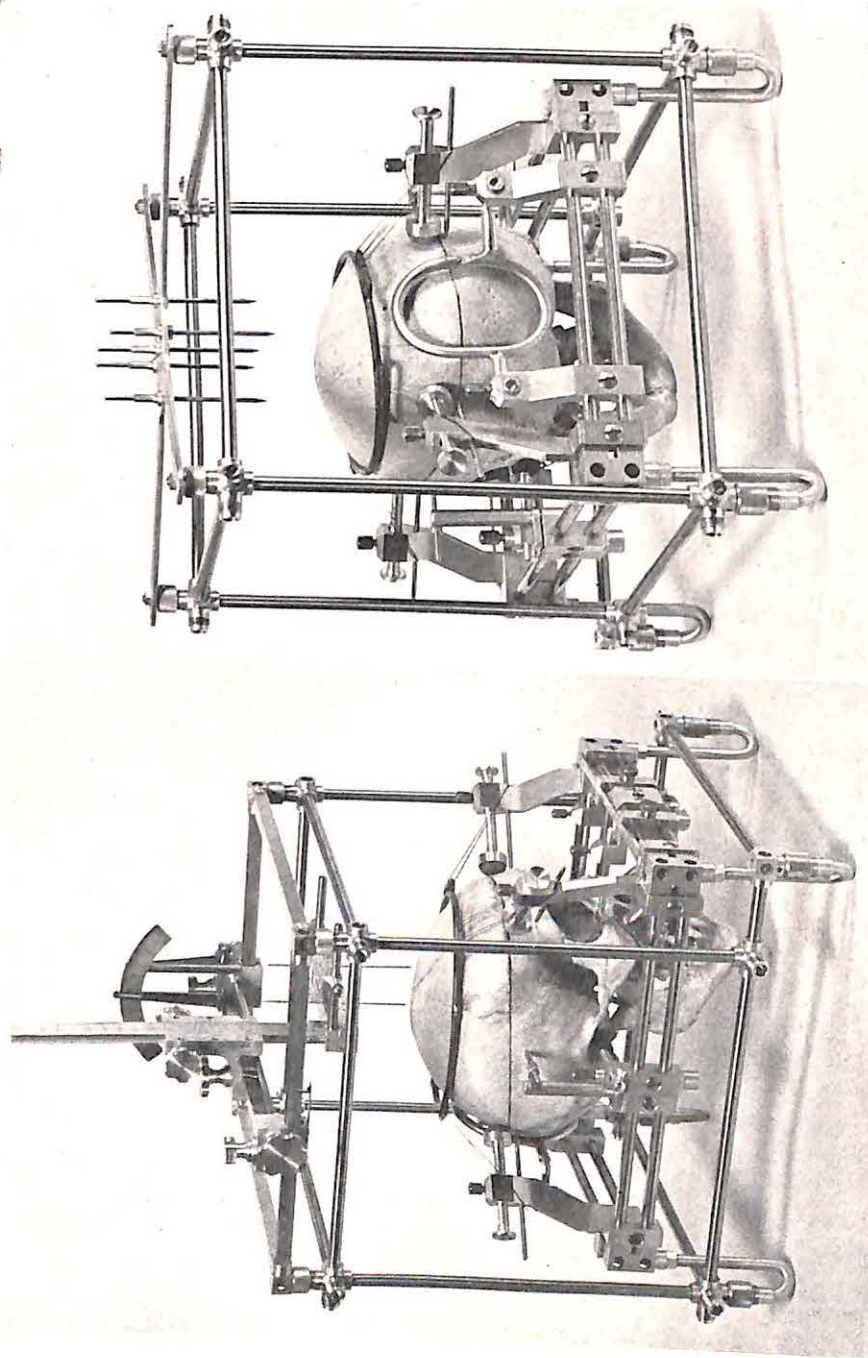


Fig. 136. The stereoccephalotome, for use in thalamotomy. (From Spiegel, Wycis and Thur: *J. Neurophysiology*, vol. 8, 1951.)

THALAMOTOMY

Thalamotomy was developed by Spiegel and Wycis.¹⁸³⁻¹⁸⁶ It is carried out with the aid of a stereotaxic apparatus developed by these authors in modification of the Horsley-Clarke apparatus (Fig. 136). These authors recommend the placing of at least five discrete lesions in the dorsomedial nucleus of the thalamus, destroying about one-seventh of that nucleus on either side of the brain (see Chapters IX and XII).

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INTEGRATION OF PHYSICAL TREATMENT WITH DYNAMIC PSYCHOTHERAPY

PREPARING THE PATIENT

Preparing the patient for any type of physical treatment should have as its primary aim helping the patient to accept, intellectually and emotionally, the need for such treatment, as well as helping him master any anxiety the idea may engender. Once a sound trustful relationship has been established between physician and patient, this aim may be effected practically by explaining to the patient in terms acceptable and comprehensible to him the general rationale of the treatment, why it is indicated in his case, and by convincing him of its necessity or desirability—for instance, that it is the only way or the quickest way for him to get well and that it will save him time and suffering.

Fortifying the patient and his relatives with foreknowledge of the treatment's side effects, probable course, and outcome is an aid in helping him and them to master the situation. It may be pointed out, for instance, that the purpose of the treatment is to bring out his own best self by restoring some measure of stability and balance to his nervous system. The findings of the Funkenstein test and the patient's own experiences during the test may be used to illustrate this point to the patient. The danger of enhancing anxiety in electroshock treatment should always be mentioned as well as the measures taken to prevent it by providing sufficient additional non-convulsive treatment. It should be emphasized that the treatment is not meant to alter his personality, but rather to help restore his real self, that it is not intended to make him think in a way alien to his true self, nor is it supposed to make him forget, although he may experience some forgetfulness as a temporary side effect, or he may have more lasting memory lapses about unpleasant things that occurred during his illness or at the time of the precipitating event.

In brief, one should be as honest and open with the patient as possible, always, however, taking into consideration the patient's problems and personality and how what is said will strike him. Before lobotomy, for example, the rationale of the procedure and the patient's individual need should be explained to all patients who are in good contact with reality. In severely disturbed patients, particularly paranoid ones, it may, however, be wise not to go into much detail but rather to adapt the preparation for the procedure to the patient's degree of understanding and to the range of what is acceptable to him.

Similarly, in insulin treatment, adequate explanation should be given regarding why it is indicated, usually based on the autonomic response pattern. Insulin is particularly acceptable to the patient after failure of electroshock treatment which had been distasteful to him because of the build-up of epinephrine-conditioned anxiety. It is then pointed out to the patient that insulin treatment has a soothing effect, particularly on anxiety.

The period immediately prior to actual treatment is also extremely important in allaying the patient's fears and anxiety and in helping him to relax. The physical surroundings and the attitudes of those dealing with the patient are probably the most important factors. The role of the nurse enters here. She should be cheerful, helpful, and comforting. In electric treatments it is much better, whenever possible, to treat patients in the intimacy of a private room rather than in one of the row of beds in a bustling out-patient ward, where the patient can hear the stentorian respiration of others under treatment. In one of my cases such an experience resulted in the patient reexperiencing her postshock anxiety for a long time after her recovery whenever she heard gurgling sounds or the cooing of pigeons on the roof outside her window.

With some patients it is also a good plan to relax them further after the initial preparations have been made for treatment, for instance after the electrodes have been fastened, by the preambles of hypnotic suggestion—rhythmically intoned suggestions to be completely at ease and relaxed, “sleeping deeply, at ease, and relaxed,” as well as a suggestion that the patient have a pleasant dream. At this point one might ask the patient about his most pleasant experience or suggest that he think of himself lying lazily on the beach with the sun glistening on the water. It is remarkable how such a suggestion may outlast or outweigh the brief amnesia that usually follows convulsive electric treatments. Not infrequently upon waking from a first electroshock treatment, a patient may report to a relative how pleasant the experience was and how he dreamed he was lying on a beach in the sunshine. Suggestion of this sort will, of course, help prevent only that anxiety and distaste that is based on the reality situation, but will have no effect in preventing the build-up of anxiety of the type that may be precipitated by enhanced sensitivity to epinephrine.

THE THERAPIST'S ROLE

The therapist must play a well defined role in any treatment situation, whether the situation is one of intensive psychotherapy or one primarily involving physical methods. This role must be dictated by the personality and needs of the individual patient. While such a role is often intuitively divined, it is desirable that it be well thought out and highly conscious in the therapist's mind, a role to be played flexibly and spontaneously according to the requirements of the immediate situation but one that follows a basic plan. The therapist can never be entirely impersonal and detached unless that role fits the needs of the patient. The doctor-patient relationship in physical treatment, as elsewhere in psychiatry, is crucial.

Not so long ago I treated a 44 year old woman who had a severe depressive illness with paranoid features in which resentment against her husband and foster mother featured prominently, and which involved severe abdominal pain and soreness that had caused considerable alarm and received much medical attention for over six years.

The patient had been brought up by an aunt who had adopted her as a young child, obviously because of a need for love rather than a need to give love, and who from the beginning had forced the patient into the position of being a diligent mother figure to her, the aunt. The onset of the patient's gastrointestinal symptoms coincided with the time her aunt and foster mother developed a serious illness in the course of which she withdrew her previously exuberant, smothering, demanding affection toward the patient, replacing it with petulant indifference. The patient then bitterly and resentfully rejected her foster mother and turned to her husband and son for solace, not too successfully, however, except for their concern about her "physical condition."

Several years later she began to show hostility toward her husband also, continuously berating him, resentfully digging up minor incidents that had not bothered her before, such as the time the car broke down on their honeymoon twenty-three years ago. Up until this time the marriage had been quite congenial. The husband, however, had taken his wife's aggressiveness meekly with only occasional short bouts of passive resistance. Subsequent exploration of his history made it obvious that his submissiveness was intimately tied up with his rearoused guilt feelings for having deserted his own mother to go with his father, at the age of 12; his mother died shortly thereafter.

The second time I saw the patient an unending stream of vituperation against her husband emanated from her. She declared she actually came in to seek treatment not for herself but for him: "I would be fine and dandy if he were well. His main motivating power is his will be done. We got along fine till two years ago, but since then I have realized he is selfish. None of his attempts not to be selfish have been sincere. He kicks people when they're down. He fires people when they cross him."

After about two hours of such projections and displacements, I rose and said, "Now look here, Mrs. X., this will get us nowhere. This could go on and on. Any point I ask you about yourself, you always answer in terms of some grievance against your husband. It seems almost as if you don't dare think about yourself, about what you are, and what your emotional needs are. Apparently they were fulfilled in some way till a few years ago, and we have to find out what went wrong. Also this thinking about his secretaries—I feel you are stretching the point and that in that respect you have gone beyond reality and are actually misinterpreting the factual situation. I am sure that if your nervous system were well, you could size up reality perfectly well. As far as your husband is concerned, we have to find out what makes him tick and what emotional needs of his you do not fill that causes this battle. On the other hand, his nervous system is a healthy one, and I think we can work as has been shown, apart from your symptoms, your nervous pains, your sleep-functioning nervous system, and then we can work out these problems."

With this patient, the role I assumed was obviously that of a kindly, sup-
porting, but firm father, possibly with the admixture of magician ("I will give you a healthy nervous system"). The patient accepted this approach docilely and rather gratefully, no doubt because one of her most basic emotional needs was to be permitted to play a really dependent role. Her foster mother had frustrated any dependent needs from early childhood on. Her weak, guilt-laden husband could not allow her too much dependence without being seriously threatened himself. So when the doctor assumed this strong paternal
thetic guidance.

Through her treatment that role was played to the hilt with all the drama that could be injected. A flexible, variegated program of combined convulsive-nonconvulsive treatments, mostly of three minutes' duration, proceeded with-

out setback, and it was possible to begin intensive insight-promoting psychotherapy after the fifteenth electric treatment, when she was ready to give up some of her projections and to accept insight into her dependent needs and their insatiable unreasonable nature. The causes of her hostilities were worked through with her as well as the ways she expressed them and the ways she avoided gratifying her dependent wishes. As she came to accept her hostility as arising from a frustrated need for love and was given paternal permission to give and accept love, she was able to give up her rigid, bristling defenses and express her softer feminine potentialities. The husband was concurrently given ventilative and insight-promoting therapy as well as helped to a greater understanding of his wife's behavior. The total treatment period extended over two months, during which the patient received twenty electric treatments of combined convulsive-nonconvulsive type and the same number of psychotherapeutic sessions, while her husband had five intensive therapeutic interviews and some twenty brief ones when he accompanied his wife. This double treatment worked out very well. The patient's health and the couple's congeniality were entirely restored and in some ways improved over what they had been at best, and the patient found herself able for the first time to be patient and nonaggressive in dealing with her 6 year old son.

TIMING OF PHYSICAL TREATMENTS—WHEN TO TREAT AND WHEN TO HOLD OFF

Once it has been decided that the patient has an illness likely to respond to one of the forms of physical treatment, it is as important to know when to withhold such treatment as it is to know when to give it and to whom.

Physical treatment should be given when a patient is at an *impasse*, when the repetitive autonomy of the patient's personalized thoughts go on unchecked by any reference to reality, and the therapist finds it impossible to get into the patient's private world in any meaningful way. Treatment should be withheld, however, when the patient's communications are meaningful and he is capable of testing reality under the therapist's guidance.

Some time ago I was called in to see a young woman, aged 28, who had recently developed an acute paranoid psychosis in which hate and hostility toward her parents was a large component. She feared that they, particularly her mother, might harm her three boys, the youngest of whom was 3 months old, the others 3 and 5 years old respectively. The present episode had begun when her husband, a soldier in the regular Army, had left for Korea two days after their third child was born. When she then went home to live with her parents, they found her very labile emotionally. She cried a great deal, was given to aggressive outbursts, was alternately loving her children and screaming at them and slapping them. She confessed to suicidal impulses and thoughts of killing her children.

When I came to the house I found her, a beautiful, well built woman, sitting with her three children clutched within her arms, fiercely protective, with her eyes flashing—much like the famous painting of Medea and her children. In spite of her demeanor, she responded immediately to a protective, sympathetic, helpful, paternal approach and willingly accompanied me to a hospital, entrusting the children to her father's care. When I saw her the next morning, she was eager to talk about her problems, to work them through, and was able to associate constructively without monotonous reverberations. She was extraordinarily responsive to psychotherapy; her defenses were down and yet she was capable of insight.

Gradually there emerged the picture of a girl extremely sensitive to rejection.

tion who had always felt on her mettle to prove herself to her parents, to a carelessly affectionate but thoughtless father whom she idolized, to a rigid puritanical mother who showed her intense ambivalence to her daughter time and again, being hostilely critical about any attempts at independence and sexual maturing. The daughter had managed to get her mother's approval and to compete with her favored younger sister by docile behavior until she was 15 when she began to assert herself more as she became aware of her mother's masked hostility. From that time whenever threatened with rejection and disapproval she developed anxious reactions, with some at first not altogether unjustified paranoid features, guilt feelings, suicidal thoughts, and marked hostility and aggression toward others, especially the parents who had deprived her. In her late teens she resorted to sexual promiscuity in order to feel accepted by someone, and finally married her present husband, a man quite far below her family's social status, when pregnant by another man.

This rich psychodynamic material poured from her and she was able to make meaningful connections. This, then, was an opportunity for psychotherapy rather than for physical treatment of the shock variety, since that would have immediately raised her defenses. I continued psychotherapy for sixteen consecutive days and she was discharged from the hospital after fourteen days. This ventilation on her part and sympathetic acceptance on mine resulted in correction of her projecting, paranoid trends, but she then began to lapse into more overt depression. She put it rather succinctly: "When I'm mean to everyone I feel fine. When I'm nice to them I feel depressed," and later, "It seems I'm punishing myself and I don't know why."

At this point, when she was at a therapeutic standstill, electric treatment was begun. In the meantime some psychotherapy with the family group was undertaken. The patient responded to several electroshock treatments with clearing of her depression, but an increase in anxiety. Combined convulsive-nonconvulsive treatments kept things in better balance, however, while relations with her family at home improved markedly. As she improved they began to show more understanding and approval of her. She relapsed slightly, however, when the doctor was out of town for a week and when her mother made some hostile remarks. In the meantime supportive psychotherapy aimed at helping her reerect effective defenses was carried on.

It is interesting, however, that the peak in her improvement—which reached the point of full recovery—coincided with the time her father stayed home more and the patient kept house for him in her mother's absence. It is quite doubtful, however, if such an improvement in the psychologic climate through the process of breaking and rebuilding—that is, of getting at and recognizing much of her usually unconscious motivation and of then being able to or if, indeed, such a change in the psychologic climate at home could have taken place without her improved capacity to deal with people.

MEANING OF PHYSICAL TREATMENTS TO PATIENTS

Various theories have been advanced concerning the significance of the meaning the patient ascribes to various physical treatments, including such symbolic concepts as death, punishment, orgasm, rebirth (Gordon,¹⁰ Hayward,¹² Dearborn,⁴ Gershman,⁹ Fisher⁵). Often the implication is that the beneficial effect of treatment is primarily due to its psychologic significance to the patient.

While the patient's subjective experiences and feelings about the treatment he undergoes are of great importance to the psychologic management of the case, I am convinced that the basic effects on mood brought about

by certain forms of physical treatment cannot be ascribed directly to any meaningful aspect of the treatment. The incontrovertible evidence is that variations in treatment technique of which the patient cannot possibly be aware bring about significantly and predictably different results. I regard it as inconceivable that the patient could differentiate between the experience of being subjected to a convulsive electroshock alone and one in which this was followed by a nonconvulsive countershock which is always given while the patient is still unconscious. Nevertheless, the psychophysiologic effect of these two forms of treatment is entirely different. The subsequent fundamental changes in mood, spontaneity, and awareness obviously must arise from the physiologic changes brought about by the treatments.

This independence between the meaning the patient ascribes to the treatment and its therapeutic effect is particularly evident in those physical methods that in themselves appear to lead to the patient's strengthening his defenses or choosing stronger ones, as is usually the case with convulsive electroshock treatment, deep-coma insulin, and lobotomy—provided, of course, that the patients are properly selected for these treatments. It would appear that the strengthening of defenses per se, regardless of their quality, enables the patient to function more effectively. A patient with a classic depression, with a characteristic autonomic curve (marked blood pressure fall on mecholyl injection, no anxiety on epinephrine injection), will recover with convulsive electric shock no matter what else is being done, electric shock in these cases working as a highly specific treatment, comparable to the action of liver in pernicious anemia. Psychotherapy may be very valuable in improving the quality and integration of the adjustive mechanisms, but the patient may actually be able to function relatively well without it, following such treatments, until the old stresses and conflicts again reduce him, or his regained balance becomes jeopardized for other unknown reasons.

We come to a different situation in the case of those physical treatments which lower the defenses, such as nonconvulsive treatment and insulin subcoma. The patient's condition following these treatments represents merely a potential, a lowered defensiveness that the therapist may put to excellent use in gaining rapport, exploring anxiety-laden conflicts, and helping the patient achieve insight. If this is not taken advantage of, however, the gains are ephemeral, for the patient cannot live forever with lowered defenses and unresolved conflicts. Life problems will force him to reerect his defenses, often in a haphazard way, and they will remain as inadequate as they were before treatment. This became strikingly evident during the war when patients on insulin subcoma and psychotherapy did very well, while for those who, because of the pressure of time, received insulin subcoma treatment alone without psychotherapy, the temporary improvement ran off like water except for a few rare stalwart souls who probably would have done as well on any regimen of rest.

The same is probably true for nonconvulsive electric treatment as well. Nonconvulsive electric stimulation treatment gives the therapist a lead for psychotherapy which if not utilized will result in the patient gaining no significant benefit, except that he will tolerate subsequent convulsive electroshock treatment better because his anxiety level will still be low.

It has been my observation that the less strikingly effective are the immediate physical effects of the treatment in itself, the greater is the im-

portance of its attendant meaningful aspects. All treatment, of course, has one extremely important meaningful aspect, based on thousands of years of medical tradition, and that is that the doctor both gives to and does something for the patient. Any treatment, even if otherwise entirely ineffective, still has that one effect, namely that it comforts the patient by giving him something, be it the lowliest placebo or the most mundane advice, and it reassures him by doing something to or for him.

Electric shock and insulin coma therapy have been demonstrated statistically to contribute a good deal above that particular influence. The results of electroshock treatment and insulin coma cannot be reproduced by the large series of cases which have been treated with methods involving otherwise indistinguishable giving and doing.

When we come to the group of treatments that by themselves are relatively ineffective, quantitatively producing about the same improvement rate as that of spontaneous remissions, as with saline infusions, for instance, we discover a very interesting activating influence in the giving and doing involved. In a group in which, for instance, 13 per cent is the expected proportion of spontaneous remissions, within the first year of illness it so happens that 32 to 52 per cent will improve among the patients for whom "something was done." Other things being equal, the giving and doing involved in otherwise ineffective remedies may add that little bit extra, an otherwise insignificant contribution toward recovery, that determines that 32 to 52 per cent of the group will recover instead of the expected 13 per cent. This fact was recently strikingly demonstrated by Funkenstein and Meade, page 278, who found in a group of patients with an expected rate of 13 per cent for immediate spontaneous recovery that 43 per cent improved when they received "injections," one subgroup receiving infusions of neutral saline having a remission rate of 52 per cent. When all members of the sample are "treated," it tends to bring about a significantly higher percentage of patients apparently responding to treatment than the expected spontaneous remission rate. The selective effect of merely doing something for the patient, while unable to increase the rate of improvement to that of specifically effective treatment (84 to 91 per cent), nevertheless seems to exert a contributing influence by helping patients hovering at the borderline of spontaneous improvement to cross the borderline into marked improvement.

This is a fact that must be kept in mind in assessing all improvements, regardless of whether they follow physical or psychologic treatment. The incidental aspect of good hospital management or a nice nurse may thus assume as much importance in hospitals in which physical treatments prevail. Such extraneous factors must always be considered.

Regardless of its primary bearing on the type of treatment, it is, however, always important to work through with the patient what he thinks and feels about a type of physical treatment before asking him to subject himself to it. This is especially true of electroshock treatment. During the course of treatment, it is valuable to continue exploring the meaning the patient ascribes to this new experience, since it often is quite revealing of his basic dynamics and since it obviously has a great deal to do with his attitude toward the treatments. During follow-up psychotherapy, discussions of what the treatment meant take a somewhat new tack in that they

are now aimed at making the resulting psychophysiological changes meaningful to the patient in terms of possible psychologic changes.

It has sometimes been suggested that electroshock treatment is a punitive experience for the patient or one involving a temporary death, and this may often fit in quite well with the dynamics of the depressive patient. Actually, however, such an interpretation is by no means a common one, and is, in fact, exceedingly rare among the relatively well preserved personalities one encounters in private practice who usually respond very favorably to electroshock treatment. The punitive aspects of the treatment have diminished markedly with modern techniques and in the conditions operating in private practice without any lessening of effectiveness; and as Hoch and Kalinowsky comment, much more painful methods have been tried without therapeutic effect.¹⁵

Whenever there are punitive fantasies associated with the treatment, it is useful to utilize them in the manner of Frieda Fromm-Reichmann⁸ for the exploration of meaningful connections.

One 34 year old woman whom I treated for a severe anxiety depression with schizophrenic features told me after her first combined convulsive-nonconvulsive treatment that she had anticipated the treatment with dread, in which fear of being punished for something she had done occurred to her after I asked her for associations. When these were explored further, she recalled that the anxious anticipation which she experienced before the treatment brought to mind the fears which she had experienced as a little girl whenever she was afraid to be caught in a lie by her father. She continued—"in a way being helpless—of course, you could get up and walk out of the room but you can't do that if you want to be dignified. I remember waiting for my father to come home and see if he were going to raise hell or not. Whenever I have something unpleasant to face I want to run out and meet it, while for my father I had to wait and see if it would happen. I could never tell him the truth except the simplest every day things. Father forbade me to take dancing and singing lessons but I went on taking them nevertheless. I wanted to dance and sing, all things he did not approve of. I had to make up a web of lies about everything I wanted to do. That is where I got that big feeling of insecurity. There is not one thing I could look back on in my childhood with joy."

These matters were worked out with her in psychotherapy and then her second combined convulsive-nonconvulsive treatment of 3 minutes' duration was administered. She was seen the following day and stated, "oddly enough I don't feel any anxiety today. These treatments do remove anxiety, there is no doubt about that . . . I also had this mental skipping, not quite grasping things at times; that seems to be gone since the last treatment . . . When I approached the first treatment it was strictly nightmares; that seems to have worn off by the time I took the second treatment. . . . I must have thought I would be screaming. It is a matter of pride. I can't start emitting any sound that I don't mean to let out . . . Outside of losing my dignity I don't mind the rest any more . . . The only difference between the first and the second treatment is that when I woke up after the second treatment I was dreaming pleasant dreams—I can't tell you what."

In a very interesting paper by Dearborn entitled, "Time Out for Death,"¹⁴ the period the patient experienced as death is actually her illness, not the

electroshock treatments. In fact, an experience of rebirth, a feeling of awakening to a new world and one full of more promise is a more frequent association of patients undergoing electric shock therapy than one involving fantasies of death. Similarly, the feeling of coming to life again, being born anew, is quite common in patients recovering from deep-coma insulin treatment and occurs in some patients following lobotomy. Such attitudes can be fostered by appropriate suggestions, especially after combined convulsive-nonconvulsive treatments. Suggestions given immediately prior to such treatments will not only be clearly remembered upon awakening from the treatment but will attain a particularly impressive quality.

A patient I was treating recently for a severe depression with marked admixture of anxiety talked to me prior to her nineteenth combined convulsive-nonconvulsive 3-minute treatment about how many more treatments would be necessary. I gave her strong reassurance that a total of twenty treatments, that is two more treatments, including the present one, would probably suffice, and repeated appropriately worded suggestions predicting complete recovery while adjusting the head band and placing the tongue protector into her mouth, combined with the usual suggestions for relaxation. She submitted to the treatment gladly, which would have been quite inconceivable if eighteen purely convulsive treatments had been administered to this very anxious woman. As she was awakening from the treatment, and before opening her eyes, and while obviously still in her mild post-treatment confusional state, the patient mumbled repeatedly "only one more treatment and I'll be well." This certainly indicates that for this particular idea presented to her just a few minutes before her treatment there was no amnesia, and that she was able to integrate the treatment meaningfully into a concept of regeneration and restoration of her health.

This feeling is also, I believe, the unconscious attitude that most psychiatrists employing electric treatments attach to their performance of them, a truly creative experience related to some of the most fundamental satisfactions in any branch of medicine.

The doctor's attitude toward the meaning of physical treatment is, of course, not unimportant, particularly as it may influence him in deciding whether and how often and to whom to give the treatment—decisions that should be made on quite objective grounds. It therefore behooves the therapist to examine his own attitudes quite carefully, the imagery and fantasies shock or insulin or lobotomy evoke in him, and the reasons for and associations to his decision in individual cases.

Frequently, especially in women—at least when the psychiatrist is a male—there is a sexual connotation. When sexual submission is a central problem in the patient's psychopathology, it may be especially difficult for the patient to permit herself to undergo treatment. In my own clinical experience I have found it very easy to persuade married women to undergo electroshock treatment; they just lie down on the couch and let it happen. The aging spinster, on the other hand, may go through a terrific battle, almost of conscience, with herself, before she will submit to the experience.

At such a juncture it is essential to understand the meaning of the patient's resistance against treatment and not to force her (or him) or to imply any sort of excessive urging. The doctor must be detached, father-

like, ready to help without ever seeming to push. When such a patient pleads, "Must I start the treatments right now?" I have found it effective to reply, "It's not I who am in a hurry. It is you who should be."

A case in point was that of a very attractive 39 year old spinster, who held a responsible executive position. She consulted me some time ago about a moderately severe depression that involved a pervading sense of failure, loss of confidence, frequent crying, with a feeling of utter dependence on those around her. Her normal personality had been that of an animated, somewhat aggressive person. She was very good looking, was greatly sought after by men, but had dissolved three engagements to three different men a short time before the date set for the wedding.

When I suggested electroshock treatment to her she was apprehensive, but after a conference with her mother she decided to go through with it. She came to the treatment room; she lay down. When following the usual relaxing procedures, the nurse placed the head band on the patient she suddenly rose, full of anxiety, declaring, "I can't go through with it." She was told she might like to come back some other time, that there was no need to do it now. She returned to the waiting room and, after a second conference with her mother, ventured back again. The story repeated itself, with the doctor showing the same patient, permissive attitude. When she came in for a third time and again repeated her performance, I asked her into the office, commenting that we should find a little bit out about what this behavior meant, that we would both look a little foolish if she emerged into the waiting room a third time.

I then inquired, "Could it be that the feeling you had when you couldn't go through with the treatment was the same feeling you had when you dissolved your engagements? Are we possibly learning something about you and these reactions that puzzle you so much from this test situation?" Her behavior was then discussed with her in the light of her fear of sexual submission to men, an entanglement she had always dreaded. Following this she quite voluntarily and happily submitted to treatment, and tolerated this and subsequent treatments very well and with excellent results.

Obsessive patients, even when severely depressed, tend to interpret the treatment as aggression, probably by projection mechanisms. These are the patients who cannot tolerate being out of control of themselves at any time, and they particularly resent what one of them termed "cutting the thread of awareness" that is implied in convulsive electric treatment. Such patients can be eased into treatment by first giving nonconvulsive treatment which has the advantage of not cutting the thread of thought—the patient is able immediately to pick up the conversation where he left off just before becoming unconscious.

When Doctor Medlicott visited me in 1949, I demonstrated to him the new form of nonconvulsive treatment I had just developed. Just before the treatment, he questioned the patient, a musician and teacher suffering from an anxiety depression, about some obscure musical work. When she awoke from her treatment several minutes later, Doctor Medlicott asked what they had just been talking about. She immediately took up her learned remarks where she had left off, although now with much greater animation, facility, and obvious enjoyment.

It is probably because of this characteristic that nonconvulsive treatment does not have any connotation of death nor does it usually have the connotation of sexual abandon or submission once the initial lying down on

the couch is accepted. Nevertheless, this type of treatment is highly effective in certain disorders.

Another item of importance is to what aspect of the treatment situation the patient himself ascribes his recovery. An Italian woman suffering a severe depression with paranoid features recovered following a series of eighteen electroshock treatments, although she had previously failed to improve following a series of twenty given elsewhere. When I saw her for her check-up visit four months after discharge, she was a picture of health, happy, beaming, wearing a tasteful new hat she had recently made herself. I asked her to what she attributed her recovery. She replied, "Doctor, that place was terrible and those treatments were terrible, but you came every day and told me, 'You will be happy, strong, and well,' so now I *am* happy, strong, and well."

During the war, in treating combat neuroses, my patients routinely received three days of deep narcotherapy, followed by two weeks of insulin subcoma accompanied and followed by individual and group psychotherapy. In asking recovered patients to what they ascribed their recovery, I received the most divergent answers. Some said, "Gee, that deep sleep, that really did it. I felt like a new man when I woke up." Others replied, "That sleep was horrible. I had the most awful dreams. But then on insulin, boy, the food started to taste good and my muscles felt bigger and stronger every day. That did it. Those talks were just the old Army game. That didn't make any difference." Others disliked the sleep and thought that the experience of gorging themselves with food while on insulin was rather revolting, "But when you explained to us what happened, that gave me the idea that I shouldn't let it bother me." Others did not think much of the talks I gave them, but thought that, "seeing those other guys get well," gave them optimism themselves.

Thus it would appear that the meaning of the treatment to the patient depends on his own personality dynamics and past experience, and on this basis some aspects of the situation may be more meaningful and significant to him than are others. This does not mean, however, that what he ascribes his recovery to is actually what got him well—although in certain cases it may be. It is instead that he appears to value most that part of the treatment that was most acceptable to him.

DYNAMICS OF THE IMMEDIATE POST-TREATMENT PHASE

Whenever there is a gradual transition from deep unconsciousness to full awareness of surroundings, as occurs following most forms of electric treatment and insulin coma treatment, the foggy state before reality testing and the usual control functions are reestablished—that is, before the ego is restored to full functioning—may be exceedingly rich in and revealing of some of the patient's most basic attitudes and wishes and often of his most pressing conflicts. In rapid succession the patient may recapitulate certain major stages in his development, indicating by the relative intensity with which he reexperiences them how important they are in his present psychic organization (Mosse,²¹ Millet and Mosse²⁰). It is in this period that the alert psychiatrist may gain valuable clues to underlying conflicts and confirmations of his clinical judgment about the patient's personality organization.

Confusion is generally a prominent feature of the patient's behavior im-

mediately after convulsive electric treatment. That this confusion is of organic origin is evidenced, among other things, by the fact that after convulsions administered with low-intensity unidirectional current this confusional period tends to be much less in both duration and depth; at times it is fleeting or practically absent. It tends, moreover, to diminish further after a number of treatments with currents of the less strong unidirectional type, especially if they are immediately followed by nonconvulsive stimulation.

Transient, and occasionally permanent, memory defects are also common sequelae of electric treatments, although they tend to be less prominent when low electrical intensities are used. After the first treatment it is not uncommon for the patient to wake up and ask, "When are you going to give me my treatment?" In subsequent treatments, however, the patient will remember a good many details such as the adjustment of the head band and the placing of the tongue protector. With a low-intensity machine such precise recollection of events immediately prior to treatment may be present from the beginning, but even in these patients certain selective forgetting takes place. Clearly in these cases in which "organic confusion" is absent this is not amnesia of the organic type but instead a process of repression ("symbolic amnesia"). It is obvious, of course, that this repression was facilitated by an organic change resulting from the passage of electric current (Holland).¹⁴

In those types of treatment that bring about a more severe and initially organic confusion, the element of repression may be obscured but is nonetheless present. A striking example of selective forgetting in these circumstances is the almost invariable occurrence of unhappily married women giving their maiden name when asked, immediately following shock, "What is your name?" Doctors Löwenbach and Stainbrook¹⁵ originally called my attention to this phenomenon in 1941, although they later published their observation—that women patients often gave their maiden name following electric shock—without reference to the psychodynamic implications; I have since used it as a routine question and found it a valid indicator of marital dissatisfaction. When the patient's marriage is a basically happy one and bears no relation to the precipitation of the illness, the patient when asked her name on awakening will always give her married name even if she is only recently married. A patient, however, whose marriage has been unhappy and has contributed to the precipitating causes of the disorder will invariably give her maiden name no matter how long she has been married, as was recently demonstrated in the case of a woman engaged in marital battle for over twenty-seven years.

Immediately following electroshock there is a raising of certain defenses, inadequate and unrealistic at first, but progressively more adequate as the ego functions are strengthened and restored. The first defense appearing on the scene is thus a blind and furious repression and denial, while the later ones are more in keeping with the ego's customary defensive operations. It is at this point that psychotherapy can be exceedingly important in helping the now strengthened ego to find new and more suitable and adequate ways of dealing with conflicting needs and the realities of the external situation.

As the patient awakes from insulin coma he may go through a brief period of anguish and fear of death or there may be much excited aggres-

sive behavior; here, as in electroshock, certain pressing problems and conflicts may emerge before the ego regains full control. This stage is immediately followed by one of childlike, trusting dependence. The doctor coming on the scene will be greeted by the patient as a good parental figure with whom he is safe.

In the phase immediately following lobotomy there is likely to be much more prolonged and deep regression, with the patient recapitulating over a period of days, sometimes weeks or months, his infantile behavior regarding eating habits, excretory training, and other important phases in his development.

One of the most prominent defensive mechanisms to appear almost immediately following lobotomy is a curious type of denial, a sort of sublime disregard for what had previously been most disturbing to the patient, although upon questioning the patient may admit that the condition, be it pain or hallucinations, still is present; now, however, it is devoid of its affective component, undoubtedly on the basis of the severed cerebral connections. While this reaction very much resembles a primitive denial, it occasionally differs in that the patient can admit the matter in question into consciousness easily and without anxiety. It is as though the recognition stayed at a preconscious level rather than at the strictly unconscious level of classical denial.

The denial, however, may be even more thorough-going and bland. It is well known that the patient who has been freed of pain will usually answer in the affirmative when asked if he still has the pain, but now it no longer matters to him. It has been undone, as it were, denied by his practice of living and isolated, as it were, from his personality. Rylander²² treated a patient, a Salvation Army Worker and the wife of a distinguished Protestant clergyman in Sweden, who for many years suffered from a severe depression with ruminating, obsessive self-torment based on the idea that she had committed sins against the Holy Ghost. After a successful lobotomy she became happy and carefree once more. When cautiously asked about the Holy Ghost she answered laughingly, "Oh, the Holy Ghost,—there is no Holy Ghost." Thus she had solved her problem quite neatly by the simple defensive operation of denial, as a child might do.

CHANGES IN DEFENSES INDUCED BY PHYSICAL TREATMENT AND MODIFIED BY PSYCHOTHERAPY

Next to the basic change in mood, a profound alteration in defenses and accessibility is the most obvious change following physical treatment, and this second change would seem to be an outgrowth of the first. It is common experience that a change in mood can bring about a remarkable difference in how one looks at life, at one's problems and how to deal with them. The cause of the mood change is irrelevant, whether the improvement comes from a good night's sleep from which one awakes cheerful and buoyant to approach life with new zest and energy, or whether it is the rise of spirits following profound relief when something one had worried about or feared actually turns out all right.

In the new mood, formerly pressing problems may seem trivial and easy to deal with; there is a new psychodynamic balance of adjustment mechanisms within the personality. The psychoanalytic formulation of the

ego now having new reserves of energy at its disposal seems a trenchant conceptualization of what has taken place.

It is imperative that during and following the course of physical treatment the therapist be aware of these shifts in psychodynamic balance as they occur so that he can intervene constructively to reinforce them or to modify them in the direction of greater flexibility, strength, and maturity. Such psychotherapeutic intervention may make the difference between mere improvement and a more thoroughgoing recovery. From my experience with such patients, particularly following electric and insulin treatment, it is my observation that they tend to return to the type of defensive operations they found most effective in the past, before their acute break. It is therefore very worth while to scrutinize with the patient his types of defensive operations and what they are directed against. Such post-treatment prophylaxis may contribute significantly to prevent relapses by helping the patient to regard old conflicts in a new light and by helping him to develop more constructive and flexible ways of reconciling his needs with the demands of reality and conscience.

Each type of physical treatment seems to be followed by a more or less specific pattern of changes in mood, defenses, and accessibility. A brief description of the specific patterns arising from electroshock, nonconvulsive electric stimulation, insulin coma, and lobotomy, together with illustrative cases, follows:

1. Convulsive Electric Treatment. In descriptive terms it may be said that following convulsive electric treatment defenses become stronger, at first in the direction of repression or projection of some of the disturbing and disrupting aspects of the conflict. In the setting of a basic change in mood, the ego functions of the personality become sufficiently strong to reerect more normal defenses against some of the most conflictful material.

A case which because of its drama and intensity was exceedingly enlightening to me about what actually went on during convulsive electric treatment was that of a 32 year old woman who consulted me in a state of marked depressive and anxious agitation with strong paranoid coloring. Until recently she had run a nursing home, an occupation for which she had no training whatsoever and which she had started on the spur of the moment without having had any professional conditioning of her attitude toward sick people.

She dated the onset of her disturbance from the time she had been anesthetized for a tooth extraction. The man she hated most, and wanted to kill, was the anesthetist who she decided had given her the incorrect anesthesia: "When I awoke after that tooth extraction I was as nutty as a fruit cake. I screamed. I wanted to kill everyone including myself, but particularly that doctor. If I could get my hands on him I'd choke him, I'd claw his eyes out, and if I had a gun I'd shoot him!" She had repeatedly called the anesthetist who had anesthetized her for the recent tooth extraction to tell him she hated him and that she was going to kill him and herself, once even calling his mother and telling her that she hated him and that he was the cause of her misery. She called the hospital authorities, the medical society, and the Governor's office, demanding that the doctor be dismissed from the hospital and his license revoked. The anesthetist subsequently told me that under the pre-operative sedatives, before the pentothal had been injected, the patient abreacted a great deal of hate and hostility toward old people, shouting that all old people should be dead. (At this point, it might be interjected that in addition to the reasons given below, the patient's violent reaction to this doctor was

undoubtedly partially a displacement of her repressed reaction to another physician who ten years previously had advised an abortion, stating that it would be "as easy as pulling a tooth," but to the contrary the patient had suffered prolonged pain and a severe infection that eventually necessitated a hysterectomy.)

It required a certain amount of courage and determination to subject this woman to electroshock treatment, especially on an out-patient basis which was the only way she was willing to be treated, but I was sufficiently confident that she would respond well and I was particularly confident in her husband's constructive and helpful attitudes, so that I was willing to go through with this treatment program.

Initially I gave her combined convulsive-nonconvulsive treatment of three minutes' duration through temporoparietal leads. This was followed by a considerable lightening of tension and hostility. After the third treatment she said, "I am coming to life. All you've got to do now is keep me from killing Doctor X."—she still regarded him as the source of all her trouble. After the twelfth treatment she stated she was greatly improved but she still felt she would kill the doctor if she saw him—however, she added good naturedly, it was not likely she would run into him and she no longer felt like going after him.

After the thirteenth treatment a remarkable thing happened. As she was lying down on the couch for the fourteenth treatment she said: "You know what I think made me ill? Running that nursing home. There were sick and old people there. I never knew old people can be so repulsive and as nutty as fruit cakes" (the same words she used to describe her own mental state). It was interesting that she no longer mentioned the doctor who presumably was the villain in the piece. She continued: "The worst thing was how awful their children were to those old people. Most of them didn't come to visit them, didn't even bring them cigarettes." She used to go out and buy cigarettes for them out of her own money because it bothered her so to see these old people so neglected.

I inquired whether she herself ever felt hateful to older people. She said, oh, she used to hate her father although she loved him dearly now. He used to beat her brutally when he was drunk, which was most of the time, and her mother offered her little protection against him. She recalled how as a child she frequently felt like killing him, choking him, clawing his eyes out (the same expressions she used in reference to the anesthetist). Now, however, she loved her father, he was sweet, and she did everything she could for him, even buying him liquor because he no longer was bad when he drank. She was also extremely solicitous about her mother's welfare; even when she was vacationing in Florida she would call her mother repeatedly to make sure everything was all right.

It became obvious that in adult life the patient had resorted to successful reaction formation against her quite understandable hostilities to her parents. Her precarious balance, however, was suddenly threatened by running a nursing home for which she had no professional orientation, where she came in by their children. When under conditions of lowered contempt and hostility dental to her anesthesia for tooth extraction—all her old hate and hostility erupted unchecked, the tremendous attendant anxiety threw her into a psychotic reaction in which the most prominent trends were projection and displacement.

As her depressive and anxious feelings lightened under the influence of electric treatments, she was gradually able to lower her rigid defenses of projection and displacement and gained more direct insight into what effect running a nursing home had had on her. In subsequent psychotherapy her

hostility against her parents was ventilated and she gained insight into its relation to her oversolicitous behavior toward them and her reaction to the neglected old people in her establishment. Her self-esteem was bolstered and she came to regard herself not as a woman with deep incomprehensible hatreds, but rather as an extremely nice woman who was good not only to her parents who had brutally maltreated her as a child but also to repulsive people in general—certainly a restoration of effective defense in the direction of her habitual reaction formation which had served her well prior to her recent illness. At the same time, however, her hostilities were made understandable, reasonable, and acceptable to her, so that she could deal with them more realistically instead of having to resort entirely to a brittle and rigid reaction formation. This successful equilibrium was well under way by the twenty-first combined electric treatment and was reinforced by several subsequent sessions of intensive psychotherapy. She remained well for five months, when she suffered a mild relapse with predominantly anxious symptomatology ("I am afraid of everything, at times I can't breathe"), which responded promptly to two nonconvulsive electric stimulation treatments under pentothal sodium anesthesia and six sessions of psychotherapy.

2. Nonconvulsive Electric Treatment. Nonconvulsive electric treatment, on the other hand, acts to permit the individual to lower certain of his defenses, particularly the most rigid and unrealistic ones such as projection, generalized inhibition, fearful overalertness, repetition-compulsion, regression, turning against the self, and the like, and the patient may then freely speak of matters hitherto forbidden and hedged round with anxiety. It would seem that the decrease in anxiety, which is almost a specific effect of this form of treatment, permits the ego to lower some of its rigid defenses against anxiety-provoking material, even when the patient has clung to these defenses for years.

Such was the case with a 42 year old married woman who had suffered from pathologic mood swings most of her life, of severe disabling degree for ten years. When I first saw her she had been, for the last five years, in a state of what might best be called irritable mania, a state in which she was extremely unstable, labile, irritable, hostile to those about her, and given to periodic deep depressions. This condition had followed in the wake of a predominantly depressive period of five years' duration.

During the initial phase of her hospitalization I treated her with nonconvulsive treatment, and under this treatment the irritable, hostile, compulsive aspects of her manic drive gradually faded, leaving behind mostly a cheerful manic animation with occasional swings into mild, sentimental depression. During this period she poured forth a great deal of material concerning her history and her feelings, some of which she had never been able to discuss, even during a two-year analysis. All this was given with great force behind her stream of talk, but yet these abreactive sessions not only relaxed her but she was able to make certain insightful connections. During this time she did not show even the usual hostility against the therapist, but instead was always highly complimentary and extremely pleased with the way things were going. She was delighted because she felt she was really herself again for the first time in eighteen years: "That's the beauty of this experience, that I can say, 'I, I' without shame for the first time!"

After the ninth nonconvulsive electric treatment, however, it became obvious that while the irritable, hostile, compulsive aspects of her manic drive were relieved, and with them her over-responsiveness to adrenergic stimuli (see Fig. 52 A), the thymergasic manic excitation itself, although changed into a friendly elated pattern, continued unabated, and with it her over-responsive-

ness to cholinergic stimuli (see Fig. 52 B). For this reason the patient was placed on a regimen of convulsive electric treatment.

By the second convulsive treatment the change in her demeanor and behavior was dramatic. Her stream of talk slowed down to a walk and she was again bristling with defenses. Gone was the easy, confidential rapport, to be replaced by a violent hostile-aggressive verbal outburst against physician and husband. Nevertheless, she was far calmer, quieter, and more relaxed in spite of her greater readiness to complain. There were no more abreactions of an insightful nature.

As the anxiety-soothing effects of nonconvulsive electric treatment waned, she was again forced to resort to the primitive defenses of denial, projection, and depersonalization. Her newly found feelings of self-approval were reversed. Thus after the fourth convulsive electroshock treatment, she railed: "I have been feeling perfectly lousy ever since you got hold of me, and I was perfectly fine before . . . I think some terrible, horrible thing has happened to me and I know nothing about it. I feel as if I had lost—at one time I had a mind. Now I don't have a mind. I am nothing any more! For the first time in my life I have nothing but hatred, and now I do nothing but hate. It is a very queer thing that has happened to me—now I can't talk to anybody. I feel a deep feeling of hate, and it is not *I* to feel hate. I won't be a partner of anything that is so full of hate. I can't explain anything [pacing up and down in agitation]. I don't know what you have been doing to me. I know something is being done to me that I don't want done. Oh, if only—I don't know what on earth—I just don't understand—I just cannot be a part of this—oh, I cannot! That is just what I don't understand."

Subsequent combined convulsive-nonconvulsive electric treatment did not bring about a more balanced response in this case, nor was such a response obtainable when subsequently convulsive electroshock treatment was combined with insulin subcoma (see Fig. 58 A). Eventually, however, this patient achieved a complete recovery on deep insulin coma treatment, which appears to be able to bring about a more balanced simultaneous alleviation of anxiety and compulsive hostile drive with its excessive adrenergic responsiveness and of thymergic disturbance with its excessive cholinergic responsiveness (see Fig. 58 B), with a more balanced lowering and re-forming of defenses. Her recovery has so far remained sustained for more than a year.

3. Insulin Coma Treatment. In insulin coma treatment a happy combination of both functions described above comes to the fore, so that there is greater ease in allowing forbidden material to emerge into consciousness and at the same time a gradually increasing tendency to use improved means of defense. This may be one reason why insulin coma treatments bring about more stable remissions in many patients (for example, as reported by Gralnick¹¹) than do the standard electrical methods.

The change in resistance against therapy that occurs during insulin treatment is most striking, as the patient adopts a naive, trusting, childlike relation toward his doctor. This improvement in the doctor-patient relationship that so facilitates the early phases of psychotherapy is mainly and primarily an effect of the psychophysiologic changes brought about by the treatment, although it is significantly aided by the psychologic implications of the situation in which the patient receives close and prolonged attention. emerges from the coma helpless and sick, and has to be comforted and fed. The reason for this conclusion is that similar situations brought about without insulin coma are far less effective therapeutically.

A highly gifted but quite disturbed schizophrenic boy was brought to me in an agitated state in which he was highly hostile toward and suspicious of his

adoptive parents, particularly his mother. He had been adopted when only a few days old and his first seven years of life had been very disruptive ones in the hands of his first adoptive mother, a seriously disturbed, probably schizophrenic woman who had given him exceedingly inconsistent treatment, swinging from complete indulgence to impulsive sadistic punishment. She died when he was 7 years old. A year later his adoptive father remarried. The new wife was a meticulous, kindly woman with whom the boy had gotten along quite well until his early teens, when he began to rebel against his parents' well-meaning but misguided attempts at molding him into their own highly conventional, rigid, middle-class way of life. Under this regime he became increasingly hostile toward his parents, could not get along with his contemporaries, became passionately interested in architecture to the exclusion of almost all other interests. Gradually his talk became rapid, stereotyped, and manneristic, and he had recurrent dreams of killing his mother. In his narcissistic state, he was quite incapable of understanding his parents' motives and of gaining any perspective on the situation in spite of psychotherapy.

On a program of insulin coma treatment, however, he made rapid strides toward gaining insight into his parents' own neurotic needs, and came to realize that their intentions were splendid and might have succeeded quite well with a more phlegmatic, less talented child. With this insight into their motives and methods, he was then able to deemphasize the hitherto extremely threatening aspects of their attempts to groom him for the role of a complacent middle-class businessman. During insulin coma treatment he was thus able to let down certain of his rigid defenses and at the same time build up more flexible, mature ones. In the subsequent three years he has been able to make a more equable, flexible adjustment as his parents too gained understanding of his needs and permitted him to seek his own gratification in his chosen art.

4. Lobotomy. The denial, isolation, and undoing mechanisms that appear so prominently following lobotomy are more fully described in the sections on the dynamics of the immediate post-treatment phase (p. 402) and in psychotherapy after psychosurgery (p. 412), as well as in Chapter XII (p. 264 ff.).

PSYCHOTHERAPY FOLLOWING PHYSICAL TREATMENT

Easing of Self-Driving Attitudes in Depressions. It is remarkable that in spite of our deep insights into the psychodynamics of depression and our recognition of the fact that self-driving, self-hating attitudes act as the mainsprings in perpetuating depressive states, depression still remains a mental illness that, once it reaches any degree of severity, can justly be classified as entirely unresponsive to psychotherapy alone. It still remains an open question whether this resistiveness to psychological influences is due to the fact that the disorder becomes deeply rooted in the organic pattern, particularly in the continually enhanced tone of the sympatho-adrenal system combined with inability of the autonomic nervous system to respond properly to sympatho-adrenergic stimuli and its tendency to over-respond to parasympathetic-cholinergic stimuli.

On the other hand, depression is certainly the illness that responds most dramatically to physical treatment, particularly convulsive electric shock. But as we know all too well, electroshock merely terminates the present attack but does not in itself prevent subsequent attacks. Moreover, it frequently leaves in its wake a phase of turmoil through which the patient, if left to his own devices, must gradually struggle. This period of turmoil, however, may be greatly eased by psychotherapy, which be-

comes imperative for all patients, with the possible exception of some in the older age group, whose previously adequate personality adjustment may now, even without psychotherapeutic support, serve as the basis for readjustment. Even in those cases, however, supportive and encouraging psychotherapy will render the period of convalescence less trying and troublesome for the patient and his relatives, with the added advantage that relapses may be discovered and treated early and promptly.

Since electroshock treatment may improve the mood level but will not alter the basic adjustment patterns of the patient, psychotherapy must take on a more specific aim when dealing with the younger age group of depressives whose previous personality patterns have not proved adequate, for the continuance of these old patterns will almost invariably result in a relapse. Following recovery from a depressive episode these patients will often remain self-drivers and self-haters. It is therefore extremely important to give such recovered patients psychotherapy in order to help them to better ways of living less marred by self-driving and self-hate, that is, to help them tone down and control their rigid, primitive super-ego demands.

When treated with these goals in mind, certain cases in which previous clinical experience would have led me to expect a relapse have not suffered the anticipated relapse. An example is a young teacher who had had annual depressions for a number of years, usually starting in the fall when he resumed teaching, when he was beset by doubts about the breadth of his intelligence and the adequacy of his teaching. All his depressions had been preceded by "a nervous striving, wanting to be better, wanting to improve myself," with the feeling that nothing he said or wrote amounted to anything. A previous course of electroshock treatments had provided only short-lived relief.

These self-driving, self-depreciating attitudes were worked through with the patient during and after administration of electroshock and the patient made an uneventful recovery. When I saw him the following November he reported with delight that he had escaped his usual fall depression that year. He said that he had adopted a new, more relaxed attitude and that with it he had actually accomplished more: "If I say to myself that I expect nothing and demand nothing of my thinking and if consequently I don't try to urge and push myself on to strive, I am much happier." With a smile he told me of a special method he used to help himself over rough spots, a magic charm he had written down and kept under the cellophane cover in his wallet: "I must expect nothing and demand nothing of my thinking." Every so often he fortified himself with it. As Whitehorn²⁸ has pointed out, manic-depressives love slogans as much as schizophrenics hate them. Several months later he reported that he was still doing well, adding, "I think I have finally found myself. Nothing is perfect, but things are going along well. I am able to handle everything, and I feel fine. I am O.K." Another year has passed since without recurrence of depression.

The extremes to which manic depressives go in their expectations of omnipotence in themselves is exemplified by the case of a surgeon who consulted me for a severe depression. He particularly reproached himself for the death of an 81 year old woman with an extensive abdominal cancer upon whom he had operated at her family's insistence. While he suc-

ceeded in removing a good deal of the tumor, she died two days later from ileus after one of the sutures of the intestine broke. He felt he should have done a better job with the sutures. To my queries, he replied that she did not suffer and that her prognosis, of course, had been a very poor one.

After his recovery from the acute depressive episode, which was spontaneous in his case and occurred after a few months' rest, travel, and rather superficial psychotherapy, he felt better about the matter and was reconciled to accepting the woman's death and no longer felt guilty about it; however, he had no insight into the almost delusional role of omnipotence he had cast for himself. His attitudes in this case were used in psychotherapy as a good illustration of his omnipotent strivings. To restore the health of an aged woman suffering from incurable extensive cancer certainly would have required superhuman, if not divine powers. That he should expect such accomplishment of himself showed how irrationally high he had set his standards for himself. The patient's realization of this was a turning point in his achieving an attitude toward himself more reconcilable with reality and one that the mere relief from his depression had not been able to bring about. Psychotherapy helped him become more tolerant of and realistic with himself, without interfering with his enjoyment of his work and general level of drive.

Sometimes it is necessary to go deeper than this and actually get at the introjected parental figures who are so disparaging and critical of the patient. This was the case with a woman who harassed herself and her long-suffering husband by constant reiteration of unrealistically high personal standards. Seven electric treatments succeeded in bringing about remission from her depression, but did not soften her excessive demands on herself and her husband. Although she was now gracefully willing to settle for less, she still felt keenly the lack of adherence to her standards. No headway was made until her assimilation of her father's often cruel insistence on his standards was worked out with her.

When her history was first taken she had described her father as a stern, honorable, upright man, and her mother as a soft, gentle woman who had bent to her husband's will. After the fifth electric shock (combined convulsive-nonconvulsive treatment) and the subsequent first purely nonconvulsive treatment, when her father was again discussed with her she said: "My father wasn't an easy person to get along with. He was very stern, cross, selfish. He provided a very good home for us, but he had a very strict way. I have been considered odd by people I have mingled with since, people who are more easy going. He insisted that our manners be up to scratch. I had it beat into me by him with words, 'Your blood is just as blue as anyone's. You have a proud heritage.' It isn't right to do that to a child such as I was."

When it was pointed out to the patient that she had been describing behavior very similar to what she had been doing to her husband and maybe to herself all these years, she conceded, "Maybe that's right, but I am not going down to my husband's mother's level. She is uncouth, untidy, ignorant."

The ground had to be gone over time and again, as the patient gradually worked out her resentment against her father and recognized her love for her mother. Later she said, "I have a gentler side that was frustrated. I thought I needed a love I wasn't getting. My brothers and sisters think I am hard and brutal. I sensed it even then. They resented me because my mother liked me more. I received no love from my father, neither did my sister. He showed no love for mother. It wasn't a happy home. He nagged incessantly, had a violent temper—he was brutal with words. I loved my mother dearly."

As these irrational standards were worked out with her, a greater identification with her mother rose to the surface, and she abreacted with a good deal of emotion; "I was afraid for so long that my real self is going to be hurt—maybe it still is going to be. I want to relax, lean on someone." It was a long, hard pull for the patient and the final results were not perfect but were a considerable improvement. When I saw her for her final check, six months after her second and last nonconvulsive electric treatment, given after a preceding course of five combined convulsive-nonconvulsive electroshock treatments, she was smiling and happy. She reported, "I am more at peace and contented. I think he (her husband) is behaving himself, but I don't care too much and don't delve too deeply. I have made an adjustment—we have to meet life as we find it. There is no other solution to bring happiness. Fighting it out won't do anything for us. I have my code and he has his. He probably won't change; I certainly won't. But we can find happiness and contentment. I find myself responding as I have not for many years to the world around me. I feel on the whole, life is good."

Psychotherapy Aimed at Improving Interpersonal Relationships and Helping Intrapsychic Maturation. Psychotherapy focussed on interpersonal relationships in an attempt to bring about greater maturity and flexibility of the personality is particularly important in treating such disorders as schizophrenia, schizoaffective psychosis, anxiety neurosis, and obsessive phobic states. Other writers have amply covered the principles, techniques, and theory involved; see, for instance, Frieda Fromm-Reichmann's excellent book on intensive psychotherapy.⁸ What I should like to emphasize here is that such work may be greatly and at times immeasurably facilitated by such physical treatments as electric shock or insulin coma.

It is unfortunate that many psychotherapists have denied themselves and their patients the assistance of the physical methods of treatment, and have insisted on doing the whole job the hard way. Equally unfortunate is the fact that physicians practicing physical treatment have all too often neglected these important psychotherapeutic measures, especially in the schizophrenic and neurotic groups.

An ambulatory schizophrenic woman whom I treated over a period of several years had been prevented from regressing further by physical treatment (principally electroshock and nonconvulsive therapy) and was able to return to her job, but she remained anxious, tense, guilt-ridden, and given to projections. An important denouement that accelerated her improvement more than had electric treatments occurred while much of her early life was being worked through with her. A key episode turned out to be the custom of her mother and grandmother to praise her extravagantly for her courage in going outdoors and exposing herself to the lightning, while they themselves hid in mortal fear during thunderstorms. While the child continued to do this in a desperate effort to get approval from some source, she sensed the hostility and dislike for her implicit in their attitude, and it stayed with her as part of her own attitude toward herself as well as in her resentment toward them. Only when she was able to work this through and abreact some of her hatred for them and herself, was she able to gain some increase in her self-esteem. She ultimately achieved a state of practically complete recovery after sixty deep insulin coma treatments and further psychotherapy.

Another young schizophrenic girl made an excellent clinical recovery after sixty-two insulin coma treatments—too excellent a recovery perhaps

since her defensive system was restored without the aid of insight psychotherapy, which she was not inclined to accept. A year later she relapsed into a mild depression with some neurotic anxiety and tension. Her depression was relieved by electroshock treatment but the anxious tension state continued unabated. As before, she was unwilling to let down in psychotherapeutic sessions, but did become much more accessible under ether by inhalation when she abreacted a good deal of hostility against her mother and freely verbalized her anxiety about not achieving the love of those who mattered to her, her fear of death, and her feelings of rejection and inferiority. These, together with her need for love and her dependent wishes, were worked out with her and remained accessible to her in her normal state. Her anxiety and tension abated; she was able to return home, and finally to work out a more congenial life plan than that prescribed by her parents. She has remained well in the subsequent two and a half years.

Physical treatment, particularly electroshock therapy, sometimes sets the stage for remarkable cures of frigidity, whether the woman has always been frigid or only so since the onset of depression. Such cures are not always spontaneous, although they occasionally are, as are some instances of improved potency in men that have been reported after insulin coma treatment²³ and lobotomy.¹³ In most cases the completion of the cure of frigidity must be brought about by psychotherapy, although the physical treatment has obviously been the initiating influence.

I generally do not regard the cure of a depressive episode in a young or middle-aged woman to be complete if frigidity still exists, provided, of course, the husband's potency is beyond reproach. In general it may be said that the ability of a man to carry on sexual intercourse for ten minutes or more, with his partner obtaining no satisfaction, is presumptive evidence of the woman's frigidity. A good many cases of pseudo-frigidity occur in women whose partners can maintain intercourse for only five minutes or less. The dividing line in my opinion seems to be at the ten minute mark. This does not mean that mature married women cannot obtain sexual satisfaction in intercourse lasting much less than ten minutes, but the early conditioning of the capacity to respond has to be brought about by much longer periods of heightened sexual stimulation early in the marriage. I therefore regard a woman as frigid only if her partner is capable of maintaining coitus for at least ten minutes, and regard the diagnosis as incontrovertible if he is capable of lasting twenty or more minutes.

At the completion of a successful series of electroshock treatments, I have found patients are often ready to discuss problems they would not mention earlier. Some patients have been frigid since their first experience with intercourse; others relate their frigidity to the birth of a child or to an operation or other stressful event, usually several years before the onset of the depression—although it may actually have been an early symptom or prodromal. It is particularly interesting that some women who have always been frigid are finally ready to do something about it after a course of successful electroshock treatments. Perhaps this is partly due to their improved defenses and lessened guilt, so that their femininity is no longer so frightening or distasteful to them and they can perform an about-face in their attitudes. Possibly also with some of these women the decrease in their hostility that occurs as the depression lightens and the world is seen

in a new light permits them to allow their husbands some enjoyment as well as themselves.

Even when the therapist cannot bring about insight in the patient regarding the meaning of her frigidity, it is sometimes possible to change the quality and balance of the defenses or motives working against sexual enjoyment. After first giving such patients a general orientation in psychosexual development and sexual techniques, often recommending Van de Velde's^{25, 26} books, I then approach the patient's individual problem on the psychotherapeutic level to aid in the improvement or formation of whatever defensive operations seem indicated. Thus a woman may be led to identify with feminine standards through an understanding and happily married social worker for instance, whether or not on the basis of reaction formation, permissiveness alleviating guilt, or identification. In a few cases I have found physical adjuvants helpful, among them methandren linguets (10 mg. three times daily after meals).

Psychotherapy after Psychosurgery, with Particular Reference to Psychotherapy of the Family Group. In a way, effective psychosurgery clears the stage of old ingrained tensions, permitting a rebirth and re-growing-up under the guidance of better parental figures, one of whom is the doctor, the other an understanding wife or sibling. Psychotherapy of this sort is essential in helping the patient develop new and healthy defenses and attitudes toward himself and others, in essence a new conception of self and of others, but at the same time it is essential that the important people in his world have increased understanding of him and do not perpetuate the old situations and reteach the patient the old ways.

The importance of the therapist's parental role was brought home to me vividly when I had to leave my practice for ten months to carry out an assignment for the Government. The patient in question had had a very difficult childhood between a neurotic, rejecting mother and an abusive, violent tempered, extremely critical father whom the patient hated and feared. He reacted with docility to his father but with defiant, unresponsive behavior to his mother, was entirely unable to get along with other children, was aggressive, unruly, developed obsessive defenses, and was given to spells of foolhardy daredeviltry to prove his omnipotence to himself and confound his terrible fears. His adolescence and early adulthood were marked by feelings of sexual inadequacy; he felt inferior even to the animals he masturbated. Later he feared intercourse with his wife, fearing he might strangle or kill her.

His periods of intense tension and anxiety finally culminated in panic attacks of horror and fear, with paranoid ideas, compulsive thoughts of killing his wife and parents, negativism, feelings of depersonalization, and complete lack of pleasure in life. He saw only a bleak, black world full of potential terror. Intensive psychotherapy in combination with various forms of physical treatment brought only temporary relief, and meanwhile his panic attacks and compulsive thoughts of killing became more urgent.

Finally, as a measure of last resort lobotomy was performed by Dr. Hannibal Hamlin, following which the patient began to show remarkable improvement. His terrors and anxieties were gone, along with his tremendous hostility, and he was able to relate to his father in a realistic way. He still remained rather irresponsible and impulsive and was notoriously lacking in social tact, but gradually under psychotherapeutic guidance his social technique began to improve. Unfortunately, at this time, two and one-half months after his lobotomy, I was called away and remained overseas ten months. During my absence the

patient made practically no further progress, but showed a great deal of impulsive, childish behavior, refusing to see another psychiatrist, and finally ended up in a truculent, extremely childish and irresponsible flight from his wife and parents (who had been concurrently undergoing psychotherapy). Fortunately, at this point when I returned I was able to calm the patient, and marked, rapidly progressing improvement started almost immediately. In the subsequent four years, psychotherapy with patient, wife, and parents have helped in developing a constructive, socially useful individual from a bedeviled, tormented one. He is now assistant manager in a small business, is able to support himself and his wife in comfort, has gradually learned how to get along with people and to temper his impulsivity.

It is most important to make the lobotomized patient again acceptable to his family, so that they may live in peace and comfort with one another, and the patient may have the benefit—than which none is greater—of their emotional acceptance. This played an important role in the recovery of a 64 year old man who had suffered from a severe manic psychosis of two years' duration and from a previous briefer manic episode two years before that. In his first manic episode not only was he aggressive and hostile to his wife and daughter, but he had inflicted severe hardships on them by giving up an excellent executive position to travel all over the country, squandering his money. After his first remission his family lived in mortal fear of a recurrence, only to have that fear confirmed by a severe second episode. It was little wonder, then, that following lobotomy they at first continued to suspect and scrutinize everything he said or did for indications of a recurrence.

Following lobotomy, numerous psychotherapeutic sessions were therefore held not only with the patient but also with his wife and occasionally his daughter. The main therapeutic design in these was to let the patient express himself in his family's presence and, by the doctor's taking him seriously and respecting his views, to encourage wife and daughter to identify with the doctor's attitude and once again give the patient the respect and regard he needed.

A similar problem was solved in like way in the case of a 32 year old schizophrenic woman, a member of a large Italian family, who for a while was difficult to reintegrate into the family setting after her successful lobotomy. Regular monthly group sessions were held with the patient and her entire family. At first, father, brother, two sisters and a sister-in-law attended, while the mother, a rather rejecting person, absented herself.

Problems in daily living and adjustment were worked through in an open forum, as it were. Incidents were brought to light in which the patient's view was right and was understood by the assembly, as, for instance, when she tried to do her best and her mother became irritated with her without reason. Even the reappearance of minor obsessive trends was interpreted to the family in terms of increased initiative, and this resulted in a greatly improved attitude. In general, a supporting attitude was promoted and the whole family became interested partners in her recovery.

Even the Rorschach test was administered with the family surrounding the patient, with the same therapeutic aim in view. At first she saw the same skeletons and anatomy replete with gruesome and bloody connotations. The family—who I trust saw happier things—were sympathetic and distressed. This performance was repeated with each visit, and when the patient finally saw her first puppy dog, butterfly, and bunny, smiles of relief spread over the faces of the assembled family. From then on they were convinced she was

well again. This new optimism finally succeeded in bringing the mother into the picture, which greatly helped the patient. When the mother volunteered that the patient was nervous only with her but was much better in company, she was helped to realize that she must give up certain demands on the patient and relax certain admonitions which company did not make on the patient. With the aid of improved family attitudes, the patient has made a remarkable social recovery and adjustment.

It is sometimes necessary to give the family real insight into the dynamics of the patient's illness and recovery. The following case may serve as an example.

A 61 year old woman was relieved of a severe ruminatory obsessive depression in which for three years she had been constantly wringing her hands and accusing herself of having caused the death of her daughter-in-law by giving her an enema during the early phase of what later proved fatal cancer—at a time when the daughter-in-law merely complained of constipation. Full of remorse over her unforgivable crime, the patient repeatedly tried to drown herself in her bath, to strangle herself with her own hands, refused food, and remained in a continued state of self-accusation. Three series of shock treatments, 13, 18, and 22—a total of 53, had only negligible and transitory effect.

Subsequently, when lobotomy was carried out by Dr. James L. Poppen, the patient made a complete and sustained recovery which manifested itself remarkably soon after the operation. Three weeks later she walked into my office, serene, happy, smiling, having already resumed her household activities and having gained 18½ pounds in weight.

In spite of this superb result, both her husband and her son were rather disturbed. When asked the reason in a separate interview, they reported that she just did not admit she was any better and that worried them. She did not seem to appreciate what had been done for her. I then asked the husband and her son how she was in their opinion. Both thought she was remarkably improved. The patient was then invited in and asked how she felt. She replied that she felt fine. I inquired, "Are you your own best self? Are you as well as you ever were?" "Oh sure." "Do you enjoy life?" "Certainly." I then remarked, "Well, your husband objects that you don't seem to feel that you are any better." "Oh well," she said, "I'm fine—but I wasn't so bad anyway, was I, Doctor?"

For therapeutic reasons the patient was not confronted with her previous behavior, but was thanked and asked to leave us for a few minutes. I then explained to her husband and son that what happened to his wife was merely common and useful way of protecting her self-esteem. Normal human beings naturally repress, isolate, and deny anything that seems at variance with their own good opinion of themselves. After all, that is necessary for healthy functioning. I presented the argument somewhat as follows:

"Now what has happened to your wife is a thing very similar to the soldier who broke down overseas, who couldn't stand the idea of his enjoying survival when his companions were killed because this assertion of his egotism gave him guilt feelings he couldn't stand. Your wife was in the same fix. When your daughter-in-law died from cancer, your wife must unconsciously have rejoiced in the idea that it was she who died from cancer and not herself. That gave her severe guilt feelings as she became dimly aware of her egotism in that situation. [It was considered better therapy at this point not to discuss the patient's possible death wishes against her daughter-in-law.] Now she's normal again and the fact that her egotism had asserted itself in rejoicing because she was not the one who had died is relegated as effectively from her

consciousness as any thoughts of guilt were absent in the soldier's mind when he was relieved every time a bomb missed him. Your wife has likewise now successfully dismissed, isolated, and denied these fleeting thoughts and to herself she is the same as she has always been. In other words, your wife is normal again. It is a test of normality that we deny and set aside what does not fit into our conscious idea of ourselves. The thing for you to do now is to do the same thing your wife has done. Forget the illness as a bad dream that has no value in reality."

Husband and son appeared delighted and considerably relieved and from then on the patient's complete normal integration into her happy and busy life as wife, mother, and grandmother continued without disturbance.

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XVI

THE ROLE OF THE NURSE DURING TREATMENT

PSYCHOLOGIC FACTORS

The psychiatric nurse plays a maternal role in the real and the fantasy life of the mentally or emotionally ill patient. Apart from all the aspects of this role which she plays in her own right, she also represents the therapist as his executive collaborator. Particularly during the early developmental phase of the therapeutic relationship, but at other periods as well, she also serves as the intermediary between patient and doctor. This latter role is sometimes a very important one, a role in which she is cast by virtue of the special type of confidence which the patient extends to her. This holds true not only for the psychiatric nurse, but for the nurse in general. Against the background of the awe-inspiring structure which the hospital, clinic, or professional office represents to the average patient, he will frequently regard the nurse as his "only friend in court." The patient is apt to regard the nurse as a person who has a good deal of inner knowledge of the mysteries and secrets of medicine, but yet is not so thoroughly indoctrinated in its traditions and conventions that she would not talk to him quite freely and give him her independent appraisal of the treatment situation. This expectancy on the part of the patient that he may count on the nurse to side unequivocally with him, rather than to act solely in the interests of abstract science and administrative policy, is helpful in putting the patient at ease in a strange hospital, clinic, or office.

Some patients who may have put on a brave front for the doctor, denying build-up of anxiety or persistence of their complaints, will unbend in conversation with the nurse and feel free to admit fears which they had been ashamed to mention to the doctor. An understanding nurse can readily dispel a good many of these fears. A cheerful woman can dispel many of the formidable aspects of the treatment room with all its instruments, gadgets, and emergency equipment (which, by the way, should as far as practicable be kept out of sight except with physician patients, for whom it may be reassuring). She will use the power of suggestion, emphasizing the pleasant aspects of the treatment, the comfortable couch, the pleasant relaxing sleep, and the refreshed feeling on awakening. She may tell of the many happy patients who successfully completed their course of treatment. At the same time she must not censure the patient for his fears, but help him dispel them in a cheerful, yet understanding and appreciative manner.

The fact that the educational, social, and economic status of the vast

majority of patients is more closely identified with that of the nurse than with that of the doctor (Ruesch⁸) may facilitate the rapid establishment of rapport between patient and nurse, a relationship which the doctor attains more slowly. It is very important that nurses be adequately trained to utilize this psychotherapeutic potential to the best advantage for the patient, and to employ it in support of the program of therapy. This highly responsible role is played even by the student nurse from the moment she comes into any direct contact with patients. During a lecture on the role of the nurse in dealing with chronic progressive neurologic disease which I have been giving annually at the Boston City Hospital for a number of years, I always ask the group of freshman nurses: "How many of you have been asked by patients—especially during night duty—what their chances are, whether they will live or die, remain crippled or get well?" Invariably, all hands go up. This experience has impressed me with the need for teaching nurses the proper attitudes toward diagnosis and prognosis so that they will adequately interpret them, as well as help to motivate the patients properly toward therapy.

SPECIFIC DUTIES

There are, of course, more specific measures of therapy, particularly physical treatment, in which the psychiatric nurse is called on to assist.

One of the major responsibilities of the nurse is the maintenance of the equipment necessary for all types of physical treatment. It is best to have a quiet room away from the routine of the office, yet easily accessible to the doctor's room in case an emergency situation should arise. If necessary, this room can be both nurse's office and treatment room. The room should be equipped with a desk, one or two chairs, files for the records of treatments, an adequate medicine cabinet, and also sterilizer and emergency equipment, such as oxygen and stimulants. There should be a bed or couch with a firm mattress, pillows, a rubber sheet, an adequate supply of linen, and one or two light woolen blankets. Running water should be close at hand. In the ideal office the treatment room should be separate from the routine examining room. This arrangement enables the doctor to continue with routine physical examinations while the patient who has been treated recuperates undisturbed. Subdued colors should be used, with soft draperies which can be drawn in order to induce quiet relaxation.

The nurse will usually see a new patient during his first visit, if the need for physical treatment is determined as a result of his examination by the doctor, her main task here being to interview the patient briefly for her records after having assisted the doctor in the physical examination. The nurse usually is in charge of making appointments for the preliminary examinations necessary before treatment. In spite of the fact that the treatments may have been thoroughly explained by the doctor, the nurse needs to be reassuring and ready to answer even the simplest questions raised by the patient. The Funkenstein test may be done in the nurse's office (with the doctor giving the intravenous injection of epinephrine), and after the test there may be an opportunity for the nurse to explain about possible treatments. The patient will thus have seen the treatment room in a natural way. The explanation of the use of any equipment can come about easily in a conversational manner. Instructions as to preparations for the first treatment are also given by the nurse. It should be

explained to the patient that fasting for about four hours before treatment is advisable, and that some member of the family or a close friend should accompany him in order to drive him home. If the treatment is compared to a brief period of anesthesia from which the patient wakes in a relaxed state, the reasons for not driving are obvious without introducing an element of fear.

Electric Forms of Physical Treatment

On the day of treatment all equipment must be ready before the patient enters the treatment room. The machine to be used for the treatment is placed on a movable table or treatment cart with the cover removed; the electrodes are properly attached to the machine. The regulator should be turned to "Sample" and the machine turned on for a few minutes in order that the controls be steady. If the machine has been subjected to cold temperature, it may take up to five minutes to stabilize the temperature of the machine.

The treatment cart should also be prepared with one shallow container of dilute saline or sodium bicarbonate solution into which the sponge-covered electrodes are placed to soak until they are needed. Two rubber airways, Guedel type, or any type of soft rubber mouth gag—airway combination to protect the patient's mouth and teeth during the seizure and yet permit adequate breathing later, should be in readiness along with a supply of soft paper tissues. Emergency equipment should be checked and readily available, though not in obvious view of the patient. If a polygraph recording is to be made during treatment, the machine should be attached to the outlet and the writing needle filled with ink, but elevated from writing contact until the proper time.

The patient's treatment record is made out for the day, and the nurse checks with the doctor concerning any preliminary medication, such as atropine or quinidine. Once the treatment room is ready, the nurse looks in the waiting room for the patient. It is the nurse's duty to see that patients are not kept waiting for treatment. Since she is usually consulted for all appointments and scheduling, it is possible to prevent any unnecessary build-up of anxiety caused by having the patient wait too long for treatment. After greeting the patient and family, the nurse asks the patient to come with her. In the treatment room, the patient is asked to sit down on a chair or on the couch in order to answer a few simple questions which will give the nurse the necessary information for her records. "Are you feeling a little better today?" will usually elicit a statement by which the amount of anxiety or depression present can be estimated. The problem of memory involvement can easily be ascertained by simply asking, "What did you do last evening?" At times during these conversations there may be a flood of information which will be valuable to the doctor. The nurse should always record these facts, and suggest to the patient that he might like to discuss them with the doctor. The patient is then invited to the doctor's office for his pretreatment interview with the doctor, after which he again turns the patient over to the nurse.

In immediate preparation for the treatment the patient should first void. He then takes off his shoes, tie, belt, and any other tight-fitting outer garment. Dentures and eyeglasses are removed and placed in a safe place. Any bulky material or sharp instruments such as pocket knives or nail

files in pockets should be removed. A weekly weight chart is kept on all patients during course of treatment. Finally, the patient is made comfortable on the couch with one pillow under his head and a blanket placed over him. The nurse then checks and records pulse, blood pressure, and temperature. The patient is now ready for the final preparations which vary with the different treatments.

Combined Convulsive-Nonconvulsive Treatment of Three Minutes' Duration, by Means of Unidirectional Current. The machine used in this treatment is the Reiter No. 2 (CW 47) with electrode attachments and rubber headstrap. These electrodes are attached to the machine in a red to red and black to black sequence. Since this treatment has a brief convulsive phase, one soft pillow is rolled and placed under the arch of the patient's back, between shoulders and hips, and a second is placed under the patient's knees. An area slightly larger than the size of the sponge-covered electrodes, centered 1 inch above the upper attachment of the scapha of the patient's ears, is covered with electrode jelly which is gently massaged into the scalp in a rotating motion. The patient's hair must be pushed back to leave the surface as free as possible. (This will be referred to as the No. 2 position paralleling the No. 2 machine.) The electrodes are then removed from the solution and attached to the patient's head in position 2 (Fig. 64 C. p. 314) by means of a rubber head band which crosses in front and locks in place. This band should be secure but not too constricting. The tip of the ear should not be allowed to come into direct contact with any part of the electrode. At this point the procedure may be rather disturbing to the patient and there is need for reassurance and encouragement from the nurse. The final attachment of the electrodes to the machine is left for the doctor.

The doctor is then shown the preliminary treatment record and notified that all is ready. He comes in and takes his stand at the patient's head in order to have access to the patient's jaw and to be able to manipulate the machine which is at his side. The nurse stands at the side to assist with treatment. The doctor talks to the patient and reassures him, checks the machine, connects electrodes, and then slips the mouth gag into the patient's mouth. The nurse stands with her hands on the patient's shoulders, her fingers extending over the head of the humerus, ready to give gentle support and restraint to the shoulders during the convulsive part of the treatment. It is not necessary to exert much force in this procedure but merely to lean forward, placing her weight on her arms and the patient's shoulders in a "spring coil" manner. The doctor begins the treatment and supports the jaw. The nurse maintains the gentle support until the convulsive part of the treatment is over, noting the position of extremities, whether flexed or extended. She also notes the time of the onset of both flushing and perspiration. As soon as the convulsive phase of the treatment is ended, she removes her hands from the patient's shoulders and continuously observes the patient's color, pulse, and respiration, communicating her observations to the doctor who is administering the nonconvulsive stimulation. The patient's mouth should be kept free from saliva and mucus; the patient's hands should be kept from coming up near the head at any phase of treatment. The patient should never touch the electrodes while treatment is in process.

Respiratory difficulty may appear at the end of the convulsion. If the

patient has apnea or prolonged muscle spasm at the end of the convulsive phase, nonconvulsive countershock stimulation will usually solve this problem, but the nurse must be ready to give artificial respiration by gently lifting the lower part of the thorax in the region of the diaphragm. There is usually very little motion once the convulsive phase is over, except for aimless motions of the hands and slight muscle twitching. If the doctor chooses to give an "end spurt" during the final few seconds of the treatment, he will tell the nurse in order that she may watch the movement of the hands more carefully. Once the treatment is over, the machine is turned off and the head strap removed. The extra pillows are removed and a blanket placed over the patient. The room is darkened slightly (not enough to obliterate color changes) and all unnecessary noise and conversation is eliminated. The patient is allowed to sleep or waken at will. The mouth gag is left in place until it is either removed by the patient or slips out. The mouth gag is often the butt of extreme anxiety or hostility reactions. Some patients may clench it for as long as twenty minutes or suck it for a similar period. The nurse now remains in attendance and checks pulse, color, respirations, blood pressure. Any unusual signs are reported to the doctor immediately. While the patient is recuperating, the nurse finishes her charting of observations during treatment. She never leaves the patient alone until he is fully oriented.

Combined Convulsive-Nonconvulsive Treatment of Two Minutes' Duration by Means of Unidirectional Current. This type of treatment varies little from the procedure used in the three-minute treatment. There are two distinct differences: (1) Reiter machine No. 1 (CW 46) is used; (2) The position of the electrodes is changed to the frontal area. The procedure for the three-minute treatment is otherwise followed, using the Reiter No. 1 (CW 46) machine, until time for the placing of the electrodes. An area a little larger than the size of the electrodes is prepared with electrode jelly at the location of the dell at the junction of the frontal and sphenoid bones, immediately behind the zygoma just above and about $\frac{1}{2}$ inch back from the outer corner of the eye (Fig. 64 A, p. 314). (This position will be referred to as the No. 1 position paralleling the No. 1 machine.) There is generally less respiratory difficulty at the end of the convulsive stage with this location of the electrodes than with the bitemporoparietal location (No. 2 position).

Convulsive Electroshock by Means of Alternating Current. The procedure is again the same in general as that outlined for the previous two types of treatment. The machine used here is the Medcraft machine model B24 ECT, or sometimes machines produced by Lektra, Rahm, and others. The electrodes are larger flat metal disks and do not require soaking in solution. They are placed in the No. 1 position and, because of the directness of the contact without the protection of sponge covering, extra care should be given to the application of the jelly. Direct dry contact will produce a skin burn. This machine has different markings and the nurse should never touch the button marked "Treat." To prepare this machine she merely turns switch to "On" position to steady the controls. This treatment has no nonconvulsive phase. The electric current is usually on for only a fraction of a second. The nurse should note and record the timing and amperage used for each treatment, as the doctor may increase or decrease them from time to time.

Electric Coma. The electric coma treatment differs from the other types of combined treatment in that it requires the attendance of two assistants. It is also useful to have one of the assistants take a respirograph recording during the treatment, since early phases of respiratory difficulty can thus be recognized before they become obvious, and hence troublesome clinically. If a respirograph tracing is to be recorded, the bellows are fastened around the lower margin of the rib cage and held secure by the chain lock. The polygraph is then turned on and the pen readied for writing by suctioning the ink and adjusted to a writing position. The vent is cleared to permit a full excursion of the record of respiratory movement.

The Reiter machine No. 3 (RC 47) is used. The electrodes are movable and are held in place and manipulated by the nurse, who sits at the head of the patient during the entire treatment. These electrodes have handles and small sponge nylon-covered tips which require soaking. The patient is prepared in the usual manner and electrode jelly is massaged along the hairline from the No. 2 position to the No. 1 position and across the forehead. The direction of motion is indicated during the treatment by the doctor and usually begins at the No. 2 position, moving in a figure "S" pattern, to No. 1, and often following the hairline upward and to the center of the forehead.

When the patient is ready, the nurse notifies the doctor and puts on a pair of rubber gloves. The doctor places the airway in the usual treatment position. She places the electrodes in the designated position and maintains a firm, even pressure during the treatment. It is necessary to keep the electrodes symmetrically placed, and when they are moved it should be done slowly and maintaining the same, firm pressure. They should never come in contact with an area which has not been lubricated with jelly.

The treatment follows the pattern of a prolonged combined convulsive-nonconvulsive treatment. The first period or convulsive stage usually lasts about 50 seconds. The doctor then adjusts the airway into a deeper position to insure an adequate air passage, and the nonconvulsive stage proceeds without interruption. The second assistant checks the patient's respirations and makes notations about changes in amperage on the respirograph recording. He also watches the aimless motions of the patient's extremities. As the treatment progresses, the nurse notes the patient's color. When there is respiratory embarrassment or cyanosis, the doctor will usually instruct the nurse to move the electrodes higher and more centrally on the forehead. When the coma becomes lighter and there is the possibility of the patient waking, the nurse is usually instructed to move the electrodes back to the No. 1 position. The length of the treatment is usually 7 to 12 minutes. When the treatment is terminated, the usual post-treatment routine is followed. The patient's respirations are checked on the polygraph and the pen elevated before taking off the bellows.

Nonconvulsive Electric Treatment. This type of treatment varies more directly from those which have a convulsive phase. There is need for only one pillow under the patient's head. Anesthesia equipment consists of two ampules of intravenous pentothal sodium (each $7\frac{1}{2}$ grains) which have been dissolved in 20 cc. of sterile distilled water and are ready for injection in a 20 cc. syringe with a 19 or 20 gauge needle. It will also be necessary to have a rubber tourniquet and sterile skin prep-sponges with a small

container of alcohol. Oxygen and suction equipment are checked, and a tracheal tube laid out, although they will only rarely be used.

The Reiter No. 2 machine (CW 47) is prepared and the electrodes are placed in the No. 2 position. During the initiation of anesthesia the doctor sits at the side of the bed and the nurse stands at the head of the bed. The doctor administers the pentothal sodium rapidly, within 23 to 30 seconds. The patient is asked to count backward from 100 while the anesthesia is being given. As the patient begins to relax, the nurse slips the mouth gag (Guedel type) into place, and attaches the electrodes to the machine. After the patient stops counting, the nurse checks the lid reflex. If it is absent, the doctor further tests the depth of anesthesia by turning on a weak (2 MA) current, while keeping the needle in the vein. While the doctor does this, the nurse must restrain the arm at the wrist. If the patient winces, more pentothal is given; if not, the needle will be withdrawn and the electrical part of the treatment begins. At this point the doctor and nurse quickly change positions and the doctor remains at the head of the patient to support the jaw and to control the machine. The nurse continues to observe pulse, color, respirations. She notifies the doctor of any sudden changes in quality or rate. These are recorded later along with the time of the onset of flushing and perspiration.

After about 3 to 5 minutes of electrical stimulation the patient may begin to waken, and the nurse or the doctor instructs the patient to raise his right hand if the current feels uncomfortable. This gives the doctor a guide to the rate at which he should gradually lower the current. During the final minutes of stimulation, the mouth gag may be removed and the patient often makes verbal comments on just how he feels. This is also the period during which there may be an abreaction, which the doctor works through in psychotherapy. Following nonconvulsive electric treatment there is usually marked relief from anxiety and no confusion or disorientation. The patient may drop off into a deep sleep which may last for over an hour.

After-care Following Electric Treatment. The recuperative phase of the treatment is equally as important as the preparation phase. An atmosphere of quiet relaxation should be maintained at all times. After the treatment and during the rest period, the nurse may arrange to have pleasant music softly played on a radio (Price, Mountney and Knoss⁷). It is best to put the machines and equipment away and to restore order in the room. As the patient wakes from any treatment which has had a convulsive phase, there is apt to be confusion and disorientation. The patient may be euphoric, perhaps show affection, or be extremely restless. If the patient should become excited, it frequently suffices to turn him over on his abdomen and allow him to fight out his excitement against the mattress, at the same time stroking his brow in a reassuring manner. Restraints are usually unnecessary. It is perhaps a wise move to place a chair with one of the extra pillows on it close to the side of the couch to prevent a possible fall. Any material which seems relative to the patient's emotional problems should be recorded. The electrode jelly is water-soluble and may be washed off gently with soap and water after the patient is awake.

If the patient has either a headache or slight nausea, this can be relieved by aspirin and hot coffee. (All patients seem to enjoy a cup of coffee or tea

as they wake up. This procedure adds to the friendliness and sociability of the atmosphere, which patients appreciate and remember when they come for their next treatment.) Patients who have had the purely nonconvulsive treatment have no confusion or disorientation but they seem to require a longer period of rest. This also allows the patient time to ask questions which he may think too trivial to ask the doctor. "How does the treatment make me act?" "Do I behave badly during treatment?" "Do I say things that I wouldn't say if I were awake?" "Why am I forgetting little things and not forgetting others?"

When the patient is sufficiently recovered, the nurse helps him dress. She usually helps patients by combing their hair and making sure that all traces of electrode jelly are removed. The doctor then returns for post-treatment psychotherapy and the nurse checks on the interval before the next appointment. She accompanies the patient out to his family in the waiting room, or the ward attendant in case of inpatients, and makes his next appointments. She suggests that his companion take his arm on the stairs, as he is still quite relaxed and may not estimate steps accurately. If necessary, she may walk to the automobile with the patient.

Insulin Subcoma

The nurse will be in charge of giving the injection, usually at 7 A.M., according to the doctor's orders. Prior to injection, she will weigh the patient, take his pulse and blood pressure and, of course, communicate all unusual findings to the doctor, delaying the injection of insulin in this event. She will keep the patient under close supervision and chart the onset of perspiration, keep a record of the pulse rate, noting the exact time of each examination. She will also chart changes in mood and mental content, both before and after the doctor's visit. At 10 A.M. she will bring the patient his high carbohydrate breakfast, preceded by glucose in orange juice if necessary.

Insulin Coma Treatment

Insulin coma treatment makes the greatest demands on the quality of nursing care, perspicacity, and thoroughness of the attending nurse. To meet these demands a good nurse should be familiar with all the facts set forth in Chapter IX and X concerning insulin treatment; i.e., she must not only be qualified to make reliable observations, but she must also understand their significance so that she can initiate or take appropriate action if the doctor happens to be occupied with an emergency situation in another patient. She is in charge of giving the injection according to the doctor's orders in the morning, after taking temperature, pulse, blood pressure, and respiration. If any of these findings impress her as unusual, she should defer the injection until consultation with the doctor. Throughout the treatment she will keep careful half-hourly records of the pulse, and she will be thoroughly familiar with the fact that a drop in pulse rate during deep coma is a danger sign and an indication for immediate interruption. It is not necessary to check the blood pressure repeatedly throughout the treatment; especially during the phases associated with hyperkinetic or hypertonic phenomena, blood pressure readings will not be reliable. However, she will immediately take the blood pressure if the appearance of pallor or cyanosis suggests impending circulatory collapse, so that the doctor can be summoned immediately and will have all the

necessary data to make his decision as soon as he answers the alarm. Whenever such phenomena indicating vascular collapse occur, the patient is usually limp and the blood pressure readings taken in such phases will be accurate. The nurse will also keep careful watch over the patient's respiration. If respiration should become disturbed owing to the accumulation of mucus or saliva, such simple maneuvers as correction of posture, raising the head and upper chest, or turning the patient on his side will usually suffice. If this does not relieve the patient, suction must be applied and the alarm given for the doctor.

During the subcoma phase of treatment, the nurse will keep in close contact with the patient, but avoid stimulating him excessively with too much conversation. If the patient complains of thirst, she should supply water (150 cc. at a time), but no other nourishment. Allowing the patient to drink water during the subcoma phase not only does not retard the onset of coma but will, in patients who go into coma slowly, accelerate the onset of the coma phase.

Perhaps one of the most responsible duties of the insulin nurse is to chart the onset of the various phases of the treatment, particularly the phases of coma. This duty is essential because it determines the length of time of treatment, which must be long enough to be effective, yet limited sufficiently to be safe. As stated previously, in the chapters referred to, unless otherwise ordered in special cases by an experienced therapist, the total period of coma must not exceed one hour, and the total length of deep (medullary phase) coma must not exceed fifteen minutes. This applies only to treatments from the fourth coma onward. The first three treatments should be much shorter, their length to be determined by the doctor (see Chapters X and XIV).

In determining the three phases of coma, namely, light, medium, and deep coma, the nurse of course must limit herself to those signs which she can competently and properly interpret. It is the responsibility of the doctor to carry out repeated examinations to determine the specialized neurologic signs enabling him to make a more precise diagnosis of the depth of the coma. The nurse, therefore, should chart only the three main stages. She must leave the diagnosis of the more detailed subdivisions of these stages, such as the various phases of thalamic and hypothalamic release, to the physician. The following are the criteria for determination of the three main phases which the nurse should observe and chart:

1. *Light Coma.* Light coma begins when the patient has slipped out of contact, i.e., when he no longer answers questions and no longer is able to carry out voluntary purposive actions. It is important to realize, however, that throughout light coma involuntary though purposive reactions, for example, reflex-like righting reactions such as corrections of uncomfortable posture, and nonpurposive mass reactions such as violent withdrawal from painful and minor irritant tactile stimuli, are not only preserved but actually enhanced.

In summary, therefore, light coma, as charted by the nurse, is a state in which the patient is out of contact but in which he will still correct uncomfortable postures and will still react or over-react to painful stimuli. The over-reaction occurs particularly during two phases of light coma, namely, thalamic and posterior hypothalamic release. In the latter phase, rage reactions may also be elicitable.

In some patients a brief intervening period may be noticed during which the patient, while no longer answering questions, may still be able to respond with some voluntary purposive reactions; this intervening period may be classified as borderline coma. The nurse must bring this matter to the doctor's attention for his decision as to whether he wishes to count this period as part of sopor or as part of the coma. It is best in the beginning to count this period as part of coma, until the subsequent progression of the phases of coma proves that it may be safe to leave the patient in coma an hour after the time at which contact is fully lost. In patients who do show this phase of borderline slipping out of contact, it never lasts longer than 15 minutes.

2. *Medium deep coma* begins when the righting reflexes are lost, i.e., when the patient no longer responds to uncomfortable positions, particularly of the head, by automatic correction. He will, however, still respond to pain by wincing; this persistent pain reflex can be readily tested by pressure on the supraorbital nerve.

3. *Deep coma* begins when response to painful stimuli, including pressure on the supraorbital nerve, no longer takes place. The physician, of course, will confirm depth of coma by examination of the pupillary light reflex which will be abolished in deep coma. The positive sign of Babinski, which may be present in light coma, is almost always present in medium deep coma, and may be present in the early phases of deep coma. The nurse will keep a careful chart of all the observations, correlated with a continuous record of the pulse rate.

The nurse is also in charge of maintaining the equipment and material for interruption, both by gavage as well as by the intravenous route. She will keep an assortment of sterile 50 cc. syringes filled with 25 per cent dextrose and fitted with sharp needles laid out and covered by sterile towels. She will keep, for the purposes of gavage and for subsequent administration of oral glucose, adequate amounts of body-warm 50 per cent glucose, as well as emesis basins, litmus paper, and stomach tubes with attached cylindric funnels fitted with a suction bulb. To the 50 per cent glucose solution to be given by stomach tube and/or subsequently by mouth after intravenous or gastric interruption, one-seventh volume of lime juice or orange juice should be added. She will also have at hand an emergency tray supplied with sterile syringes and needles together with the assortment of drugs discussed in Chapter X, as well as all equipment and supplies for the interruption of irreversible coma as described on page 163 ff.

Carbon Dioxide Therapy

In preparation for administration of carbon dioxide therapy, the nurse should be responsible for the assembly of the apparatus and further nursing aids, including an emergency stimulant tray, emesis basin, Guedel-type rubber airway, and an adequate supply of blankets. The importance of pretreatment procedure and post-treatment care as outlined previously must be borne in mind. The patient should fast for three hours prior to treatment; he should void before treatment is initiated; his tie, belt, and the like should be loosened, his glasses put aside, and dentures, gum, or any other foreign objects removed from his mouth. The nurse must record the patient's pulse, blood pressure, and any pertinent com-

ments on his mood. The CO₂ tanks should be checked beforehand in order to assure that they are in working order, but in such a way as not to alarm the patient with a loud or forceful expulsion of gas from the tanks. At this time, the emergency pure oxygen apparatus should also be examined and placed in an easily accessible position ready for immediate use if necessary.

During carbon dioxide treatment, the nurse will regulate the flow of carbon dioxide mixture and pure carbon dioxide from the tanks, while the doctor, after proper reassurance, will hold the mask on the patient's face and ask him to take deep regular breaths. The doctor will count the breaths in a loud voice. The patient is instructed with each count to raise his right arm as a signal that he has heard the count. Two tanks, one containing 25 per cent carbon dioxide in oxygen, the other pure carbon dioxide, are joined by a Y connection to a tube leading to the mask. The nurse will first open the tank containing carbon dioxide in oxygen, letting the gas escape at an approximate rate of 15 liters per minute, while keeping the pure carbon dioxide tank closed. After the patient has taken five or six breaths, the doctor will signal the nurse to feed some pure carbon dioxide into the mixture by gradually throttling the 25 per cent mixture down to an approximate flow of 4 liters, and gradually adding increasing amounts of pure CO₂, beginning at an estimated rate of 6 liters per minute and increasing the flow to approximately 15 liters per minute. After five or six breaths of this more concentrated mixture, the doctor will signal the nurse to supply only pure CO₂, whereupon she will shut off the tank containing the 25 per cent mixture and allow only pure CO₂ to flow, at an estimated rate of 20 liters per minute. After six to eighteen breaths of pure CO₂, the patient will be in coma, whereupon the doctor will remove the mask and the nurse will shut off the supply of CO₂.

If the coma is a quiet one, this ends the participation of the nurse in the actual treatment. If agitation is connected with coma, she will assist the doctor in turning the patient on his stomach so that his aggressive agitation will be vented against mattress and pillows rather than against therapist and nurse. Flurries of excitement with this form of treatment are usually only of very short duration. After the patient relaxes, the doctor will encourage him to tell his carbon dioxide dream and will work this and related material through in a psychotherapeutic session. At the conclusion of this session, the patient will be left in the nurse's care until he is ready for departure.

Intravenous Ether Drip Infusion Treatment

Before treatment is begun, the nurse will have recorded the patient's pulse, blood pressure, and temperature. The patient should void before treatment, and should be comfortably dressed, with all constricting clothing loosened. The blood pressure apparatus remains on one arm throughout the course of treatment. The other arm should be strapped to an arm board. The intravenous equipment and the sterile solution of 1000 cc. of 5 per cent glucose in saline should be prepared by chilling overnight prior to treatment in order to prevent evaporation of the 25 cc. of ether, which is to be added immediately before the treatment and thoroughly mixed (see p. 374). The nurse will also have on hand sterile syringes, needles, tourniquet, and an emergency stimulant tray. Pulse and blood pressure

are checked during the course of the treatment at a time interval specified by the doctor, and in addition on appearance of any unusual symptoms, such as pallor. The nurse must frequently check the intravenous solution for suspension and shake it gently at least every fifteen minutes, as well as continuously check the rate of flow. If the patient falls asleep during the course of treatment, the nurse must rouse him from time to time to prevent him from falling into too deep a sleep. If the patient complains of dizziness, the rate of flow should be decreased. In the event that the needle slips out of the vein, the nurse must change the infusion to the other arm, as the ether is irritating to the tissue. When removing the needle at the close of the treatment, she must take care to leave 50 cc. of solution in the bottle so as to prevent ether from entering the vein in too strong a solution. The patient's temperature should be taken one hour after treatment is begun, and at the close of treatment. The patient should be covered warmly during the course of intravenous ether administration. For the occasional patient who displays apprehension, it is the nurse's duty to offer reassurance, and to note those changes in mood or abreaction material which will supply valuable information to the attending physician.

Ether Abreaction Treatment by Inhalation

Prior to the administration of ether by inhalation, the patient should fast for five hours. The patient should void before treatment begins; he should be comfortably attired and all tight clothing loosened, glasses removed, dentures, gum, or other foreign objects removed from the mouth. The nurse will record the patient's pulse, blood pressure, and temperature, as well as mood changes or unusual signs or symptoms which she may consider important. She will prepare the patient by applying cold cream or petroleum jelly liberally to his face, and by putting a drop of mineral oil in each eye to protect the conjunctiva. She will secure a towel about the patient's hair, and another around his neck to protect hair and clothing. Before treatment is begun, she will cover each eye with a small wad of absorbent cotton.

The nurse will arrange the anesthesia equipment for the anesthetist, check and have on hand emergency oxygen equipment, stimulant tray, and emesis basin and, during the course of the treatment, check the patient's pulse, respirations, and color. If the patient reacts to treatment with flurries of excitement, the nurse will restrain him gently. She will follow through carefully after treatment with the suggestions outlined in the introductory section of this chapter.

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TREATMENT OF ALCOHOLISM

ALCOHOL ADDICTION

Psychotherapeutic Aspects. The treatment of alcohol addiction is one of the most difficult tasks in psychotherapy. The problem is twofold, first because of the prevailing social attitude toward alcohol, and second because alcoholism, especially in individuals with a depressive or paranoid personality predisposition, represents one of the most profound and stubborn manifestations of the death wish, which in spite of its ambivalence is nevertheless of extraordinary strength. The social attitude provides the cloak of rationalization which buttresses the defenses. To attack such profound death wishes on the psychotherapeutic level requires an all-out mobilization of the positive forces of life and health, of all that is opposed to passivity and death. Certain types of psychotherapy and certain personalities among psychotherapists, as well as certain settings, are more adapted to this particular task than are others.

In discussing the treatment of any illness it is worth while to consider those conditions under which spontaneous improvements or cures take place. The spontaneous cures of alcoholism are frequently associated with some form of religious conversion experience through which the patient is successfully transformed into a crusader for temperance and abstinence.

This conversion experience is occasionally aided and reinforced by a delirious episode, a phenomenon which is reminiscent of the way organic confusion states play a role in the treatment of other personality disorders, for instance, the function of the organic confusion incidental to electroshock or insulin coma in alleviating endogenous psychoses. The delirious episode in alcoholism is helpful, however, only if the patient has in some way been prepared for the religious-conversion experience.

A striking example of this is an ex-alcoholic who became a successful preacher in Durham, North Carolina. In the course of the seven years following his conversion he built a church and did outstanding work in rehabilitating alcoholics among the inmates of the county workhouse, where I first came in contact with him. Impressed with his success, I invited him to present his case history and an account of his cure at a clinic on alcoholism which I was giving at the Duke University Medical School on May 6, 1942. He presented his case magnificently. He described how for many years he had been a drunk, how he had been kicked off the police force because of his heavy drinking, and how after trying his hand at cab driving for a while he had finally sunk to the gutter. During this period he had several fearful and harrassing episodes of delirium tremens: "The fear and horrible sights!

You try to seek peace and find none—you still see the hideous sights—every horror imaginable to the human mind.” In his then unregenerate state he did not understand that God sends delirium tremens to the sinner who drinks to warn him of what is in store for him if he does not find the way to God. His first inkling that God could deliver him came from a talk with a former alcoholic in February, 1935. This man told him about God and God’s love for mankind: “He began to tell me how little I knew about God and His goodness. He told me about the eternal life. He explained in a simple way that you must believe in God and He will deliver you.”

The patient felt, however, that he had sunk so low there was no hope for him; he continued drinking. Soon he became more tremulous than he had ever been. It was then that the thought first occurred to him that something strange was about to happen to him because the Lord had said, “Thou shalt tremble.” Shortly thereafter he had his most horrifying attack of delirium tremens. Great black beasts with bloody snouts rushed at him. Weird reptiles spouting fire, giant, prehistoric creatures with slimy scales bore down upon him while fire and smoke erupted through the floor all around him. At the climax two giant snakes coiled themselves about him and began crushing him. As he was held in a vise between the two monstrous snakes, in terror he shrieked aloud to God for help. Suddenly all the horror and darkness vanished; he was surrounded by an ethereal light and Jesus, the Son of God, stood before him in all serenity and peace. Christ told him that if he followed Him and rooted drink out of his life, he would be saved. “When I saw the Son of God before me I knew that drink was gone from that day. He put in my heart a love that will hate the evil. God gave me a love in my heart that can hate that which would destroy me.” From then on our patient knew that the Lord could save all others who came to Him; and he made it his mission to bring this message to others suffering as he had suffered.

There is no doubt that after the setting had been prepared for this man a delirious episode made up of both a horrible and an ecstatic part played a great role in his cure through conversion, and became the motivating power behind the successful crusade he carried on throughout a considerable part of North Carolina.

It is interesting to note that the cofounder of a far more successful and widespread movement in the treatment of alcoholism—namely, Alcoholics Anonymous—acquired his strong motivation from a similar ecstatic experience in the terminal phase of a delirious episode, in this case also after a previous contact with another former alcoholic who had undergone religious conversion, although this contact was not directly and immediately effective.⁴⁴ Mr. William W. describes how in the course of heavy drinking he fell into a black depression for which he was hospitalized. In the agony of his suffering, he made a frantic appeal to God:

“The result was instant, electric, beyond description. The place lit up, blinding white. I knew only ecstasy and seemed on a mountain. A great wind blew, enveloping and permeating me. It was not of air, but of Spirit. Blazing, came the tremendous thought, ‘You are a free man!’ Then ecstasy subsided. Still on the bed, I was now in another world of consciousness which was suffused by a Presence. One with the Universe, a great peace stole over me and I thought, ‘So this is the God of the preachers: this is the Great Reality.’ But reason returned, my modern education took over. Obviously I had gone crazy. I became terribly frightened.

“Dr. Silkworth came in to hear my trembling account of the phenomenon. He assured me I was not mad; that I had perhaps undergone an experience

which might solve my problem. . . If he had said 'hallucination' I might now be dead. To him I shall be eternally grateful."

In comparing the two accounts of experiences, there is no doubt that the first was a full-blown case of delirium tremens terminated by an ecstatic visionary hallucination. The account of the second case speaks of a depression which was suddenly relieved by an ecstatic experience which may be definitely characterized as at least part of a delirium tremens—Mr. W. speaks of being terribly frightened, of feeling that he had gone crazy, and refers to his trembling. Obviously Mr. W.'s delirium was of a milder type in which partial insight was preserved, although insight was by no means lacking in the first man's more classic delirious experience.

In both cases the delirious experience had been preceded by preparation for religious conversion with the expectation of a miracle. It is interesting that in the first man's case previous experiences of delirium prior to the contact with his converted friend had produced no such reaction nor had they terminated in a comparable ecstatic visionary experience. The delirium in both these cases served to reinforce the "change."

Such cases are probably more numerous than we realize; for example, the reformation of the alcoholic prototype in that melodrama of American folklore—"Ten Nights in a Barroom"—is likewise mediated by a delirium which followed the crushing episode of his daughter's death which was directly connected with his drinking.

In all these cases there follows then a release of energy frequently directed at converting others, a stimulation and directedness of positive and constructive pursuits of life which afford a marked contrast to the passive and self-destructive pattern prevailing prior to the conversion experience.

It is, of course, important to realize that the delirium per se does not produce such a profound change in the living pattern of a patient who has suffered from it, but that it is capable of being utilized in that direction much as other abreactive or stimulating techniques are utilized in the right setting and if the patient is prepared for the meaningful aspects of the experience.

In the same clinic in 1942 I presented a patient who had been by no means disturbed by his delirious episodes, which he found on the contrary rather pleasant. Everything that he saw was "awfully pretty; everything was flowery and beautiful." He saw the March of Time several times "absolutely free and most interesting." He also saw himself in the newsreels saving his grandmother's life by carrying her out of a burning house. Another alcoholic had the time of his life riding on a fast-moving streetcar, hitting and smashing all automobiles in his way. It is obvious that these patients were still unprepared for the meaningful experience which the delirious episode provided to the "prepared" patients described above.

It is of course true that stimulation to growth is possibly the most important ingredient of all psychotherapy, being the antipode to passivity and death. But possibly because of the deep personality disturbance characterized by most profound death wishes, an extreme degree of such stimulation appears to be called for in treating alcoholism. It is an old empiric fact that the strongest stimulation to change, that of the evangelistic type, seems to be more successful in alcoholism than other more standard forms of therapy, and likewise, that this form of stimulation,

which includes integration into a new group, is far more successful in alcoholism than in any other type of emotional disorders.

Among the most complete and permanent cures of chronic alcoholism are those in which the patient has been successfully transformed into a crusader for temperance and abstinence (Alexander²). It is therefore not surprising that at present the most potent force in the field of group therapy of alcoholism is the movement known as "Alcoholics Anonymous." William W., one of the cofounders,⁴⁴ as well as other speakers for the group, declared that there must be a profound shaking of the individual's pride in himself and a recognition of his complete failure and defeat, that he must have been divested of his pride and rationalizations. If this does not come about spontaneously by his having sunk as low as the gutter, or otherwise having hit bottom, deflationary techniques are to be used. William W. emphasizes the importance of what he calls the crushing of "prideful obstinacy," and that "marked religious conversion experiences come mostly to individuals who know complete defeat." He adds: "For drunks the obvious answer was deflation at depth" . . . "it followed that ego deflation must also go deep or else there couldn't be any fundamental release. Apparently religious practice would not touch the alcoholic until his underlying situation was made ready." The horrors of disease, death and insanity should be thoroughly dwelt on: "We leaned heavily on Doctor Jung's statement how hopeless the condition could be and then poured that devastating dose into every drunk within range." He describes the experience of the reformed drunk, once the groundwork of deflation has been accomplished, as basically the same as that of religious conversion with its radical alteration of motivation that had the capacity of changing "utterly defeated people."

Other significant factors brought into play simultaneously and subsequently in this lay movement are identification with the group, the subsequently aroused mimesis, and later reinforcement of the new experiencing by carrying the message to others. The new experiences and attitudes have been formulated by the Alcoholics Anonymous into the following "twelve steps":

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. We came to believe that a Power greater than ourselves could restore us to sanity.
3. We made a decision to turn our will and our lives over to the care of God *As We Understood Him*.
4. We made a searching and fearless moral inventory of ourselves.
5. We admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. We were entirely ready to have God remove all these defects of character.
7. We humbly asked Him to remove our shortcomings.
8. We made a list of all persons we had harmed, and became willing to make amends to them all.
9. We made direct amends to such people wherever possible, except when to do so would injure them or others.
10. We continued to take personal inventory and when we were wrong promptly admitted it.
11. We sought through prayer and meditation to improve our conscious

contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.

12. Having had a spiritual experience as the result of these steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs."⁴⁴

The way in which the psychotherapeutic setting of Alcoholics Anonymous is successful has been analyzed in an important study by Gray, Rizzo, and Tartakoff, "Social Maturation through Membership in Alcoholics Anonymous."¹⁸ They point out that in addition to the "twelve steps" there are the four slogans, the 24-hour rule, and the AA prayer, all essential to AA functioning. The four slogans are: (1) First things first. (2) Easy does it. (3) Live and let live. (4) It's the first drink that gets you drunk. The 24-hour rule advises the alcoholic to deal with each day as it comes, "neither worrying about the past nor anticipating the future." The AA prayer is: "God grant me the serenity to accept those things I cannot change, the courage to change those things I can, and the wisdom to know the difference." All are excellent mental hygiene.

The authors also throw valuable light on how changes in personality come about, as well as on certain aspects of the conditions under which these changes occur. They point out that in most cases in which membership in Alcoholics Anonymous has been successful in producing long-term sobriety, the patient, after having "hit bottom" (defined as "a painful loss of prestige and status"), finds in AA complete acceptance, warmth, and emotional support, achieved without stirring up guilt feelings. With this support and acceptance by others with whom he can easily identify, the patient finds it possible to recognize and deal with certain aspects of his personality into which he previously had little or no insight—his 'impulsivity, his unrealistic reactions, the disruptive states of mind that had previously led to drinking. "Most of the members refer to these states as 'dry drunks' and designate resentment as the prime emotion involved. . . . A minority of the members states that self-pity and guilt feelings are the main disturbing emotions. . . . AA's agree that in order to prevent the progression of the 'dry drunk' into actual drinking, recognition of its existence is necessary as well as the need to deal with the emotions involved in a new way. This new way of handling emotions involves a gradual catharsis and ventilation, followed either by an active working out of the problem, or in some cases by a more passive method of handing over insoluble problems to a higher power." These gains are solidified in "twelfth-step work" which gives additional self-confidence and incentives to sobriety, as well as on a much deeper level allowing the patient to become the good parent. In this emotional setting the patient can allow himself to learn to live more realistically and maturely. The authors summarize the deeper dynamics of this group experience in the following words: "In coming to AA in the helpless state of having hit 'bottom,' they come, essentially as infants, to be taken care of by a mother as they have many times previously in ending a drinking spree. In AA, however, there is a fundamental difference in the type of mother figure they have chosen. They are now accepted genuinely and sincerely as worth-while people, are not criticized for their past, and are promised real and consistent human relationships in exchange for their alcohol. The sequence of events seems to constitute a corrective emotional experience for them. It starts

the process of correcting a misinterpretation of reality that they have carried throughout their lives, that is, that they are doomed to rejection, that all relationships are as insecure as their own early relationships with their mothers."

It is clear from Gray and his coworkers that there is real correlation between what goes on in Alcoholics Anonymous and the established principles of psychotherapy (Simmel³³). It is interesting to note the importance of the preliminary deflationary measure which either the patient achieves himself by "hitting bottom" or has been fostered by such procedures as William W. describes as crushing of "prideful obstinacy." Such preliminary breaking-down or deflating procedures are of importance in psychotherapy generally, irrespective of whether they are carried out by insight psychotherapy, by drug-induced narcosis, or other mental or physical means (Sargant,³² Alexander¹⁰).

The other and subsequent strong psychotherapeutic means used in this group setting are strengthening of the ego through support, acceptance, ventilation, identification, and mimesis. These are, of course, tools which the psychotherapist uses in addition to interpretation and aid to insight. In Pavlovian terms, alcoholics seem to be people with strong but unbalanced nervous systems, who must first be broken and deflated before they can be lifted up and built up.

Many members of Alcoholics Anonymous find it useful to have their treatment supplemented by more specific psychotherapeutic techniques not available in the lay group. For some alcoholics the combination of AA membership and psychotherapy is advisable, although it is by no means necessary for all. On the other hand, it is almost always helpful and a valuable supplement to a psychiatric treatment program to refer certain alcoholics under psychotherapy to Alcoholics Anonymous simultaneously or as soon as the patient is considered ready for such a step.

In each case one must evaluate the best therapy in terms of the dynamics of the particular patient. Among those exceptional cases which should not be referred to Alcoholics Anonymous are patients whose defenses would be too abruptly threatened by exposure to the group. The same is also true for some individuals whose ego would be excessively deflated by the therapist's equating them with a group with whom they could not successfully identify. The psychiatrist must judge how much group support the patient needs and how much group identification he can stand, beyond the support and identification provided in individual psychotherapy. When the identification of the patient with the therapist is based on certain of his notions of the imperturbability, masculinity, independence, achievement, and intellectual leadership of the therapist it would be unwise to throw the patient into a group toward which he may remain ambivalent.

On the other hand, when identification is difficult for the patient because the therapist may by his very reputation and personal qualities set too high an ego standard, or when identification is difficult because of differences in social-intellectual status, it may be very valuable for the patient to be given an alternative or additional anchor by solid identification with people whom, while in some ways regarding them as parental figures, he may nevertheless more readily accept as his peers.

Somatic Treatment. Some patients are obviously too sick to be helped

by psychotherapeutic measures alone, even in a setting of hospitalization. In such cases, somatic techniques are helpful, the most useful of which are electroshock and other electrostimulation techniques (Alexander¹¹), and the so-called conditioning technique.^{15, 23, 24, 26, 36, 37, 38, 42, 43}

Electroshock should be limited to those patients in whom depression is a significant element in the causation of alcoholism—which is indeed a significant number of cases. In a number of cases, chronic depression antedates alcoholism by several years. Nonconvulsive electric stimulation is advisable in those rarer cases in which anxiety-tension states are an important predisposing factor. Once depression or anxiety has been relieved by such physical means, to which recently carbon dioxide treatment has been added (see Chapter XIV), the patient then becomes much more accessible to psychotherapy.

Conditioned aversion treatments of alcoholism, particularly those using apomorphine, have been in sporadic use for some time. The disadvantage of apomorphine, however, is its sedative effect. Voegtlin⁴² introduced a non-sedative medication for this purpose that was a mixture of emetine, pilocarpine, and ephedrine. The main purpose of the addition of pilocarpine is that it produces sweating shortly before the nauseating effect of emetine sets in, thus giving the therapist a cue for the right time to offer the first alcoholic beverage. Ephedrine is added to counteract the danger of syncope. If the latter occurs, atropine may be used as an additional antidote.

For purposes of treatment, the prescription is made up in 40 cc. ampules from which individual doses may be drawn; the proportions are as follows: emetine hydrochloride, 50 grains; pilocarpine, 25 grains; ephedrine sulfate, 23 grains; water up to 40 cc. For each individual treatment, 6 to 12 minims is injected hypodermically. Two to eight minutes later the patient starts to perspire; at this moment he should be exposed to the sight, taste, and smell of alcoholic beverages—which, with proper timing, seem to produce nausea and vomiting, apparently in response to the liquor.

In order to produce the "conditioning effect," care should be taken that the liquor is offered several seconds before the onset of nausea. If there is an error in timing and the liquor is given prematurely, the onset of nausea may be hastened by administering 1 or 2 grains of emetine in whiskey.

Once nausea has set in, all types of liquor are forced on the patient, making certain that each empty glass is smelled deeply. Warm water is given frequently in order to afford easy emesis and to avoid retching. After the patient has vomited, the routine is repeated until the nauseating effect of the drug has begun to wane. The patient's stomach is then thoroughly emptied and washed with warm water, using a stomach tube. The patient is then allowed to rest comfortably, wrapped in warm blankets, until the pilocarpine diaphoresis stops.

This treatment should be given daily for a week, then on alternate days during the following week. For the remaining half year, one treatment per month followed by two quarterly treatments during the second half year is advisable.

Contraindications to treatment are cardiovascular renal disease, hepatic cirrhosis with or without esophageal varices, hernia, active peptic ulcer, or history of recent hematemesis, and active psychosis.⁴²

This so-called conditioning technique is particularly useful in obsessive

personalities subject to the formation of ritual-like habits. It thus can be used in helping the patient form new defenses. It is also useful in depressive personalities, when given during the period of heightened suggestibility after the completion of the electroshock series. Sargant³² regards the "conditioning treatment" as another example of preliminary "weakening" or deflating techniques. He states: "Repeated vomiting and physical debilitation in the conditioned aversion treatment of alcoholism often seems to lay the patient open to suggestion and psychotherapy, which would never be acceptable without it." I have recently reviewed the results of treatment in my private practice. I found that good results were obtained in about 40 per cent of all cases of alcoholism which I treated, irrespective of the methods used—that is, whether psychotherapy alone was given or psychotherapy aided by electroshock treatment, or by conditioning treatment, or by both. This does not mean, of course, that each of these patients in these various treatment groups would necessarily have done equally well in one of the other groups. The selection of type of treatment for the individual patient is an important feature.

Patients in whom chronic depression played a role and who responded to treatment would not, I am sure, have done equally well if electroshock had not been used. By the same token, I am sure the anxious and obsessive types would have done poorly had they been given electroshock; most of these did not show a sufficient amount of overt anxiety to make nonconvulsive treatment desirable. Electroshock was, of course, never the only treatment used. After the depression had been lifted by electroshock these patients were given intensive psychotherapy, which was of decisive importance in making their recovery from depression meaningful to them and in consolidating the improvement. The same follow-up psychotherapy played an important role in the treatment of patients who received conditioning treatment (which was given by another physician at my request while the patient continued in psychotherapy with me).

The intensity and type of treatment I have given to various patients has varied considerably. Among the sustained four-year cures with which I have remained in touch there is at one end of the scale a patient who received psychotherapy alone, for only seventeen sessions (this case is reviewed in reference¹⁰). At the other end of the scale is the multifactorial treatment of a man whom I started to treat in January, 1946, at first with electroshock, then conditioning treatment, followed in turn by intensive psychotherapy. He has maintained sobriety for over five years. Evidence that the original diagnosis of a significant depressive factor was correct may be found in the fact that three years ago he consulted me about what appeared to be a definite acute depressive episode which subsided after three electroshock treatments followed by a few sessions of psychotherapy. Recently I also treated his sister for an endogenous depression not complicated by alcoholism.

In summary, I feel that electroshock or other physical methods of treatment are warranted whenever it is considered desirable to alter by physical means the prevailing state of mentation and emotion. This is particularly indicated in the presence of depression, which is known to be difficult to modify by psychotherapeutic means alone.

In view of the complexity of the problem which alcoholism presents, a complex, multifactorial treatment method may hold more promise than

standardized treatment according to a cut-and-dried pattern not sufficiently adapted to individual features in each patient.

Antabuse. Recently a new tool of physical treatment has become available, namely Antabuse medication.^{17, 19, 20} Antabuse is the trade name for tetraethylthiuram disulfide, which combined with alcohol forms acetaldehyde in the circulatory system. This combination in the blood stream produces symptoms which involve chiefly the circulatory and respiratory systems, and in the proper proportions are felt as extremely uncomfortable and distressing by the patient. The Antabuse must be taken from three to forty-eight hours before ingestion of alcohol to produce this low tolerance, and the intensity of symptoms then depends on the amount of alcohol taken.

The first symptom, which appears 7 to 12 minutes after drinking alcohol, is usually a feeling of heat in the face followed by intense flushing. The scleral vessels dilate "making the person look bull-eyed" (Hald and Jacobsen¹⁹). Sometimes palpitations and slight dyspnea occur; pulse rate rises to 120 or 140; blood pressure is unaffected or somewhat lowered; cardiac output increases about 50 per cent in resting patients and 5 per cent to 15 per cent in mobile subjects (Jacobsen and Martensen-Larsen²⁰). Thirty to 60 minutes after ingestion of alcohol, nausea may set in; facial flush is replaced by pallor, and the blood pressure falls considerably. Pulsating headache usually accompanies the syndrome. The reaction will persist as long as there is alcohol in the blood stream. The patient is left feeling tired and weary but after a few hours of sleep may feel completely restored.

Great precautions must be taken before deciding to treat a patient with Antabuse, since all its effects on the human body are not yet known. Careful physical and laboratory examinations are necessary to determine the patient's physical condition before treatment and should be rechecked at intervals during treatment. Antabuse medication is contraindicated in the following conditions (Glud¹⁷): (1) Myocardial failure or coronary disease, (2) cirrhosis of the liver or any condition in which the patient has less than 85 per cent of normal liver function, (3) chronic or acute nephritis, (4) epilepsy, (5) goiter, (6) pregnancy, (7) drug addiction, (8) treatment with paraldehyde, (9) diabetes mellitus, (10) asthma, and (11) disturbance of the hematopoietic system.

Antabuse must be given with the patient's full knowledge and cooperation. It should never be given to a person who is still on a drinking spree, but is best started after at least a week or so of sobriety. Glud recommends the following dosage schedule: 2 gm. the first day, 1.5 gm. the second day, 1 gm. the third day, and 0.75 gm. per day for the next four days, always in the morning. It is advisable to try the patient with alcohol on the fourth and on the eighth days, with 40 to 50 cc. of whiskey (or the alcoholic equivalent in another beverage) on the first trial, and 30 to 40 cc. of whiskey on the second. This should be done under carefully controlled conditions, if possible, in a hospital where the patient may stay overnight.

Glud continues: "From the eighth day onward, an effort is made to adjust the dosage to that amount which, upon the drinking of 10 to 20 cc. of whiskey, produces a slight flushing around the head, a slight increase of pulse rate, and a mild dyspnea, lasting approximately 15 to 20 minutes. The daily amount of 'Antabuse' necessary to produce such a reaction will vary from one patient to another; the range of dosage is from 0.125 gram

to 1 gram. Four to six trials with this smaller dose of alcohol (10 to 20 cc. of whiskey) over a period of four to five weeks should be made to determine the maintenance dose of 'Antabuse.' This dose may then be given daily for an indefinite period."

There is danger of acetaldehyde poisoning if the dosage of Antabuse is too high and/or if too much alcohol is taken. The symptoms are breathlessness, decreased pulse rate, ataxia, unconsciousness, and occasionally convulsions. These symptoms may be terminated by prompt administration of coramine and carbogen (95 per cent oxygen and 5 per cent carbon dioxide) or pure oxygen. The disadvantage of this form of treatment, as compared to other forms of conditioned aversion therapy, is that it does not allow the patient to play as active a role subjectively and objectively. In Antabuse treatment the patient retains objective and subjective dependence on his continued daily use of the medication. Another disturbing feature is the fact that the threatening and subjectively dangerous symptoms which occur when the patient drinks after ingesting Antabuse may constitute a powerful temptation to his masochism and unconscious suicidal trends.

I have recently seen a patient who succumbed to this temptation in a manner most alarming to the physician who administered the Antabuse and to myself when I was asked to see him. The clinical picture which resulted from an afternoon's continued debauch in spiteful and what appeared superficially to be bravado-like disregard of increasingly alarming warning symptoms was suggestive of an acute encephalo-myelitic involvement, obviously due to acetaldehyde poisoning, in which cerebral edema with tentorial pressure cone leading to compression of the third cranial nerve played a part. One of the neurologic disturbances in this case consisted of selective involvement of the innervation of the internal muscles of one eye, the pupil being fixed in maximal mydriasis.

On the other hand, those who use the method have reported that it was possible to induce sobriety in patients who had not been accessible by any other method, including individual psychotherapy and participation in Alcoholics Anonymous. There is no doubt, however, that even Antabuse treatment can at best be only a preliminary or supplemental step to facilitate psychotherapy.

Adrenal Cortical Extract. A recent form of treatment based on certain as yet unconfirmed observations concerning a disturbance of metabolism in alcoholics is that of reducing craving for alcohol and increasing stress resistance in general by administering adrenal cortical extract over a period of several months (Tintera and Lovell,⁴¹ Thimann,³⁹). The dosage recommended (Tintera and Lovell,⁴¹) is as follows: "Intravenous injection of adrenal cortical extract, 10 cc. each, three times during the first day; two injections of 10 cc. each the following day, then single injections of 5 to 10 cc. daily in the following three days; in the next three weeks, intramuscular injections of from 2 to 5 cc. twice weekly with the same dosage continued at weekly intervals 'for an indefinite period.'" Hospital treatment is highly recommended but not always essential; however, the most acutely ill patients seldom need more than five days while some require only three. They recommend that during this period of hormone treatment the patient be placed on a diet high in fat, moderate in protein, and restricted in carbohydrate.

ACUTE ALCOHOLISM

The most useful drug in the treatment of severe acute alcoholic intoxication is amphetamine sulfate (benzedrine sulfate). In alcoholic coma 40 mg. or more may be given intravenously. Its effectiveness is due to its central stimulating effect (Alles and Prinzmetal,¹² Reifenstein and Davidoff²⁰), which counteracts the narcosis produced by alcohol in a way similar to its resuscitating action in other drug-induced comas, such as those produced by barbiturates or opiates.

In addition to its central resuscitating effect, amphetamine sulfate also delays absorption of alcohol (Rinkel and Myerson,^{30, 31}), probably by inhibiting the absorption from the alimentary tract and by delaying the emptying time of the stomach. Therefore, amphetamine should not be administered while the patient continues to drink, since the intoxicating effect of the previously imbibed alcohol is not dispelled but merely delayed. If taken after the bout, however, amphetamine not only has a sobering effect but, when taken the morning after, also prevents or aborts the usual symptoms of hang-over (Bloomberg¹³). In this way the drug can be used to break the vicious cycle which becomes established when the morning hang-over renews the patient's desire to drink. If used in this manner to cut into the reverberating chain of addiction, amphetamine sulfate can become a useful adjunct to psychotherapy in extramural treatment of alcoholism (Bloomberg¹³).

A more recent method of treating the stage of acute alcoholic intoxication is that involving administration of adrenal cortical extract (ACE) in the dosage as outlined previously (p. 439) (Tintera and Lovell,⁴¹ Thimann³⁹). Smith³⁴ has found that ACTH also is effective in treating the acute stage of alcoholism, but that on the whole the action of ACE is more relaxing and is to be preferred with women who have not yet reached the menopause. In his experimental set-up, Smith gave 25 mg. of ACTH intramuscularly every six hours during the treatment period or 10 cc. of ACE intravenously every six hours.

An older remedy for the treatment of acute alcoholic intoxication was apomorphine hydrochloride. Five to 10 mg. given subcutaneously has a pronounced central stimulating effect and soon produces vomiting. After vomiting has taken place and the excitatory phase of its action has subsided, there is a certain amount of narcosis similar to that of morphine. For these reasons apomorphine was employed for a great many years as a treatment for acute alcoholism. It restored the circulation and respiration of the cyanotic, comatose drunk who was breathing shallowly; it then made him vomit out what yet unabsorbed alcohol he had in his stomach and subsequently quieted him conveniently for the ward personnel. The unpleasant aspects of the vomiting episode were sometimes utilized as a form of deterrent conditioning and probably became the model for the conditioning treatment with the aid of emetine, although emetine is superior for this purpose, since it is not followed by narcosis.

All too often jail is the only place available for the night's treatment of acute severe alcoholism. It might be far more constructive to provide a special emergency ward for alcoholics so that, instead of the next morning's appearance in court, the patient could be visited by a psychiatrist, a social worker, and a representative of Alcoholics Anonymous.

CHRONIC ALCOHOLISM

Apart from symptomatic treatment, which includes proper hospital facilities, nursing, and nutrition, and among which a very important feature is that no restraints be used in alcoholic psychoses (Moore²⁸), the treatment of these conditions is essentially one of adequate vitamin medication. The beneficial effect of adequate vitamin medication is quite striking in severe alcoholic psychoses, especially delirium tremens and Korsakoff's psychosis, and still more specifically in the encephalopathy due to nicotinic acid deficiency, and in Wernicke's disease, which is due to a particularly severe and acute deficiency of vitamin B₁.

The specific beneficial effect of thiamine chloride in Wernicke's disease, particularly as far as the characteristic bilateral ophthalmoplegia is concerned, was recently confirmed by Spillane³⁵ and by deWardener and Lennox¹⁴ in nonalcoholic cases observed in prisoner-of-war camps, in confirmation of my earlier findings in animal experiments and in chronic alcoholic patients.^{1, 2, 3, 4, 5, 6, 7, 8} It is, however, important to realize that most cases of Wernicke's disease are complicated by other deficiencies of the vitamin B complex, just as conversely nicotinic acid deficiency is frequently complicated by Wernicke symptoms indicative of thiamine-chloride deficiency. In a case of Wernicke's disease in a prisoner of war which Spillane³⁵ treated by thiamine chloride alone, the patient recovered from acute generalized edema, amnesia, confusion, bilateral ptosis, ophthalmoplegia, bilateral facial paralysis, and stupor; but on recovering from this acute episode was left with "bilateral retrobulbar neuritis, nerve deafness, ataxia, and symmetrical peripheral neuritis. The latter condition did not respond to thiamine."

It is therefore important for practical purposes of treatment never to use any one vitamin alone, although one particular vitamin deficiency may be clinically the most outstanding. It is my general custom to treat patients with alcoholic psychoses by intramuscular injections of 100 mg. of thiamine chloride and 100 mg. of niacin twice daily, supplemented by a daily intramuscular injection of a multivitamin preparation such as Lyo B-C (Sharp & Dohme) or Berocca-C (Hoffman-LaRoche). In cases of Wernicke's disease, I prescribe thiamine chloride, up to 200 mg. intramuscularly twice daily; niacin, 100 mg. intramuscularly twice daily; and Lyo B-C, one ampule once a day intramuscularly. In cases of encephalopathy due to nicotinic acid deficiency, I give the following dosages: niacin, up to 200 mg. intramuscularly twice daily; thiamine chloride, 100 mg. intramuscularly twice daily; and one ampule of Lyo B-C intramuscularly.

Intravenous injection of adrenal cortical extract (10 cc. every six hours) has been found helpful in some cases of Korsakoff's psychosis (Smith³⁴), while ACTH is not. Smith³⁴ found that ACTH (25 mg. intramuscularly every six hours) was "strikingly beneficial" in treating delirium tremens; improvement became clinically noticeable within three to ten hours in contrast to the forty-eight to seventy-two required with the usual treatments. Smith believes that the action of ACTH may be accounted for by the theory that there is adrenal exhaustion in delirium tremens, a view for which he presents suggestive experimental evidence.

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XVIII

TREATMENT OF OTHER STATES OF INTOXICATION OF EXTERNAL ORIGIN

Intoxication caused by chemical substances other than alcohol may occur accidentally, due to occupational exposure, in the course of attempted suicide or homicide, or due to addiction. The treatment consists of three phases: treatment of the acute intoxication, treatment of the late after-effects of the poisoning, and, in those cases in which ingestion was due to addiction, therapy similar to that outlined in the preceding chapter for chronic alcoholism.

By way of introduction to the following chapter, it should be emphasized that the traditional clinical signs of organic psychosis—disorientation, memory disturbances, and the other aspects of organic confusion such as clouding of consciousness, slowing of reactions to external stimuli, the deliriod character of the hallucinations, and the presence of confabulations—are not always reliable differential diagnostic criteria. Not infrequently, hallucinatory and paranoid reactions occur in drug-induced psychoses which are clinically indistinguishable from those observed in schizoaffective states. Severe anxiety may frequently obscure or, on the other hand, give the appearance of clouding of the sensorium. Bewilderment, motor restlessness, and semistuporous states occur in toxic as well as in schizophrenic or schizoaffective psychoses. For example, as Moore and Gray⁴⁶ have pointed out, the effects of carbon monoxide inhalation may resemble the behavior patterns of hebephrenic schizophrenia, or may simulate dementia paralytica. Or, as Seymour⁶¹ has noted, chronic barbiturate poisoning may resemble manic-depressive psychosis. Careful physical, psychiatric, and toxicologic study is therefore indicated in all cases. The psychiatrist and physician must have thorough familiarity with those types of mental states that may be produced or simulated by drugs and other exogenous toxins.

In the following, a brief review of the treatment of the more common intoxications with presenting symptoms and signs of mental disturbance will be given.

HYPNOTIC AND SEDATIVE DRUGS

1. Barbiturates. Fully developed acute barbiturate intoxication is characterized by extreme lethargy or a comatose state. A phase of excitement, delirium, and hallucinations may precede the depressant effect of the drug. Excluding alcohol, chronic use of the barbituric acid compounds constitutes

the most common type of drug intoxication. Chronic addiction gives rise to more prominent mental symptoms than acute barbiturate poisoning (Danneman¹⁴); the latter commonly leads rapidly to extreme depression of the nervous system.

Acute Poisoning. Treatment of acute barbiturate poisoning depends upon the depth of narcosis; in the absence of coma, supportive therapy will usually suffice. In the presence of coma, emergency treatment is essential.

The most important need of the patient in deep barbiturate coma is for oxygen. Because of the profound interference of the barbiturates with dehydrogenase activity of cells, particularly those of the nervous system and the heart muscle (Brazier and Finesinger⁹), as well as the inadequacy

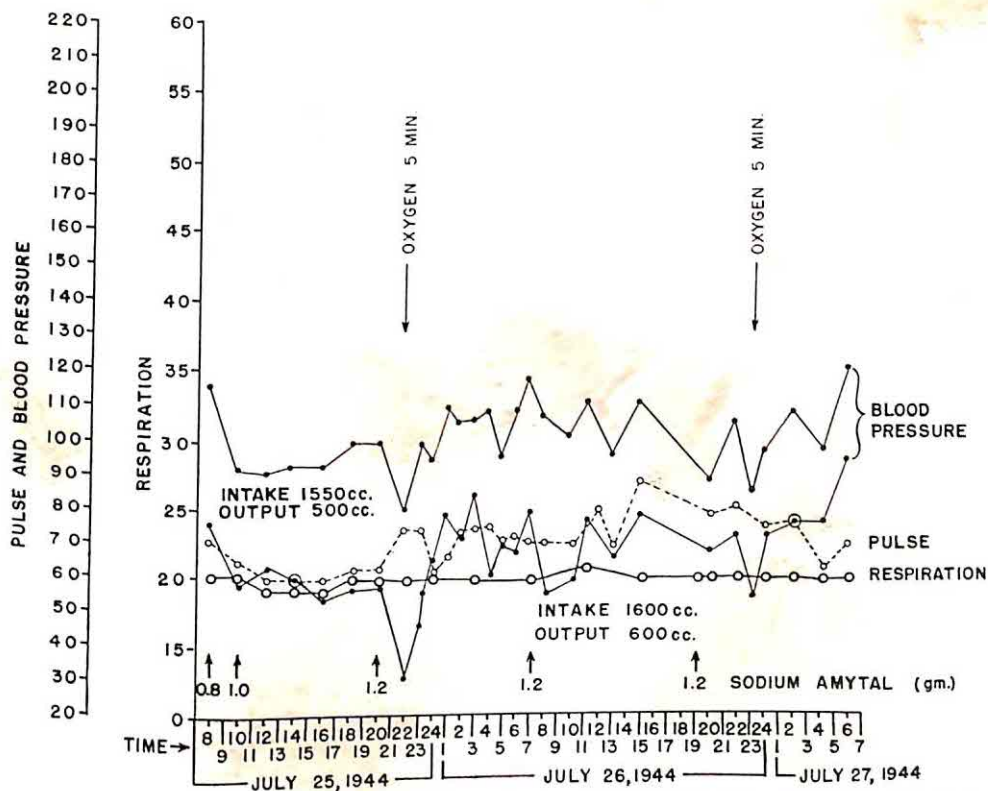


Fig. 137. Prolonged therapeutic barbiturate narcosis. Male, age 25 years, suffering from acute reactive anxiety state due to combat stress. Note that fall of blood pressure at end of first and second day of treatment to 80/30 and 82/50 mm. Hg respectively was promptly relieved by administration of oxygen by inhalation.

of the respiration under prolonged barbiturate narcosis, profound oxygen lack may manifest itself fairly early during severe states of intoxication. This anoxia not only interferes with the maintenance of cerebral functions but with the maintenance of the functions of the heart as well. It is very likely that deaths from barbiturate intoxication occurring during the first twenty-four hours are deaths from anoxia of the heart muscle, apart from depression of the cerebral vasomotor centers. It is for both these reasons that the blood pressure falls during severe barbiturate intoxication, sometimes at an alarming and life-threatening rate. Oxygen by inhalation administered in time will restore the falling blood pressure to its former level with remarkable promptness. This can be strikingly demonstrated if

patients are closely watched during states of therapeutic barbiturate intoxication administered for the purpose of continued narcosis in the treatment of acute reactive anxiety states (see Figs. 137 and 138).

Another type of reaction to oxygen lack, primarily due to shallowness of respiration caused by profound hypotonia of the musculature, including the respiratory musculature, is one in which the blood pressure as well as the temperature rises (Fig. 139). In this emergency, oxygen by inhalation

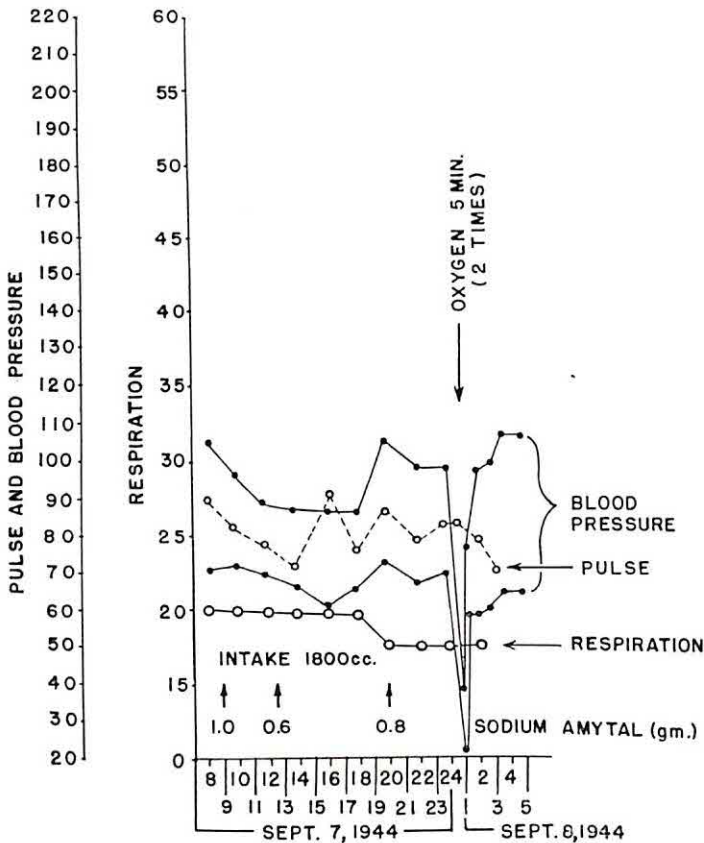


Fig. 138. Record of third day of prolonged barbiturate narcosis treatment of a 21 year old male patient suffering from combat stress neurosis. Note that marked fall of blood pressure at end of day, to 35/22 mm. Hg, was promptly relieved by inhalation of oxygen.

alone will not be sufficient, and medication aimed at restoring the muscular tone must be applied, preferably 1 to 2 mg. of strychnine administered hypodermically, to be repeated if necessary (Fig. 139).

The best analeptic for the stimulation of the activity of the central nervous system in barbiturate intoxication is amphetamine given by vein. Forty milligrams administered intravenously will waken patients in deep barbiturate narcosis within a few seconds (Alexander, Winston, and Berman⁵). Electroencephalograms show that within 10 seconds of the injection of amphetamine, the slow activity of deep barbiturate narcosis (see Fig. 140) may be replaced by the fast activity prevalent in light barbiturate narcosis (Fig. 141), although clinically by that time, the patient has already awakened fully. Eight minutes later the electroencephalogram

is usually fully restored to its normal waking rhythm (Fig. 142). Coramine or metrazol given intravenously has a similar effect (Kirstein⁴⁰).

Picrotoxin has also been recommended; however, in view of the fact that it may produce toxic symptoms (convulsions) which may require readministration of barbiturates as an antidote, I prefer not to use it.

Robie⁵⁵ recommended nonconvulsive electric stimulation therapy as a helpful analeptic procedure in deep barbiturate coma; the usefulness of

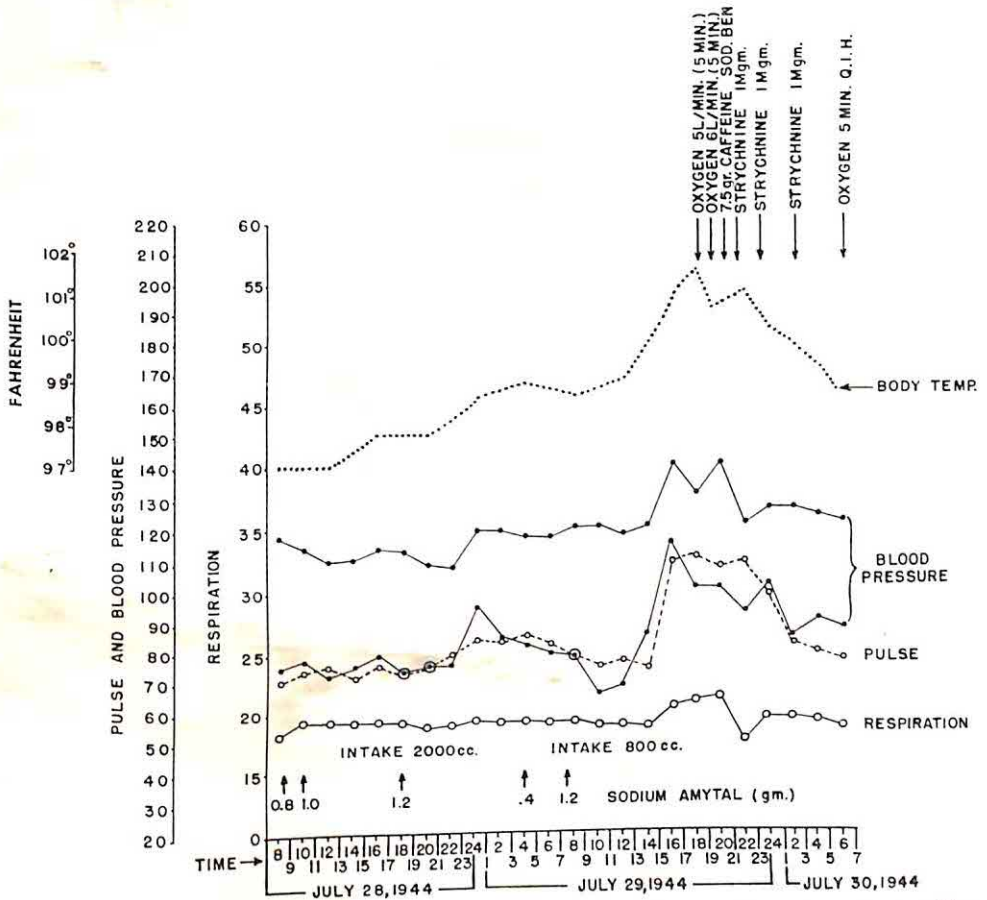
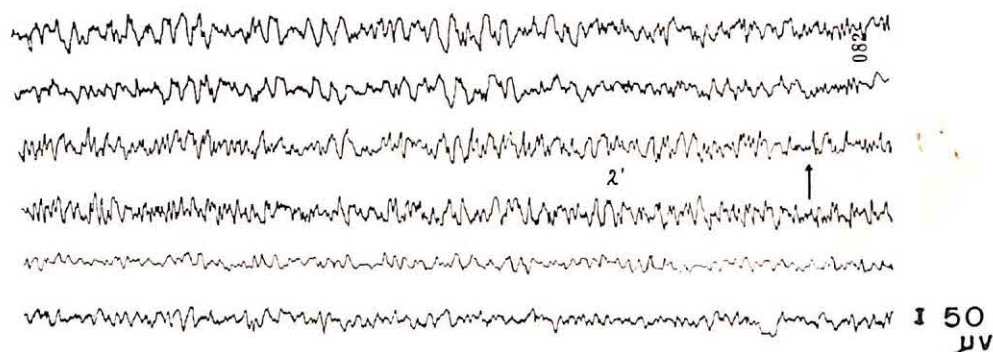


Fig. 139. Prolonged barbiturate narcosis treatment of a 22 year old male patient suffering from combat stress neurosis. Note rise of temperature, blood pressure, and pulse rate on the afternoon of the second treatment day associated with shallowness and increased frequency of respiration caused by profound hypotonia of the musculature, including the respiratory musculature, relieved by hypodermic administration of strychnine.

this measure was confirmed by Furst and Reiter²² and others. Sufficiently large comparable statistical series treated with and without electric stimulation respectively have as yet not been published.

Gastric lavage (provided the gag and cough reflexes have not been abolished, or that other measures have been taken to prevent aspiration) and the intravenous administration of glucose in saline to combat dehydration are additional measures to be employed when indicated.

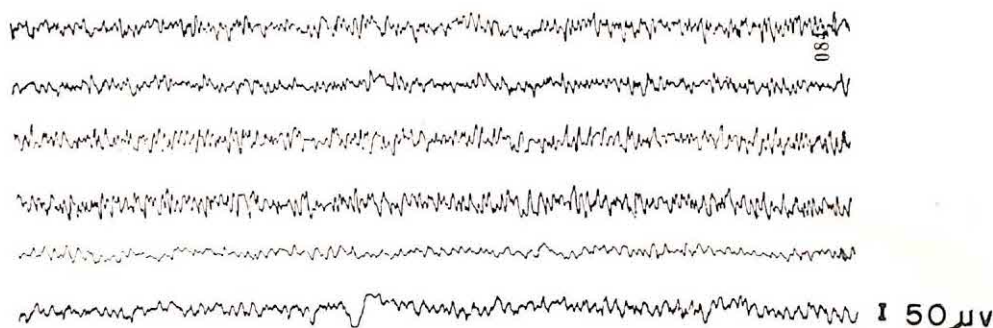
Chronic Poisoning. Delirious syndromes or manic states usually with confusion as well as disorientation, speech disturbances, gait disturbances, tremor, and absence of reflexes may follow chronic barbiturate intoxica-



1 Sec.

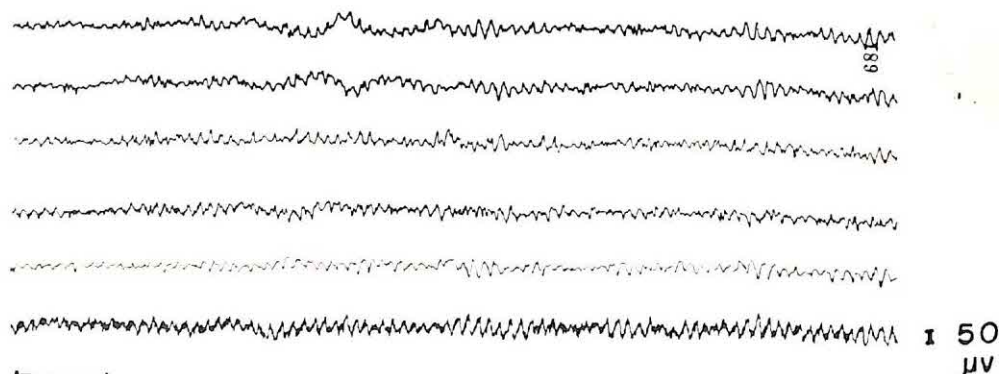
Figs. 140-142. Electroencephalograms during deep pentothal sodium narcosis and after resuscitation by intravenous injection of 40 mg. of amphetamine sulfate (from the data of Alexander, Winston, and Berman⁵).

Fig. 140. Deep narcosis, 2 minutes after completion of pentothal sodium injection (0.2 gm). Note slow high voltage waves. The arrow on the right marks the beginning of the amphetamine injection.



1 Sec.

Fig. 141. Patient fully awake, 10 seconds after intravenous injection of 40 mg. of amphetamine sulfate. Note fast cerebral electroactivity, similar to that seen in light barbiturate narcosis.



1 Sec.

Fig. 142. Normal cerebral electroactivity, 8 minutes after waking and completion of amphetamine injection.

tion. As Seymour⁶¹ has pointed out, some cases of barbiturate poisoning may resemble manic-depressive psychosis. Such psychoses are more commonly observed in patients with a history of emotional instability. Chronic addiction is often the result of the addict's effort at self-medication for symptoms of neurosis or chronic depression. Barbiturates are also commonly used in conjunction with or as a substitute for alcohol; the alcoholic may resort to barbiturates to counteract his alcoholism, then to alcohol to counteract the depressant effects of the barbiturate, thus establishing the vicious cycle of events typical of such addiction, which can be broken only by withdrawal and total abstinence. Hambourger,²⁶ Alexander, Moore, and Leary⁴ and Moore, Alexander and Ipsen⁴⁵ have pointed out the growing and alarming popularity of the barbiturates as the method for suicidal attempts.

Mental symptoms in cases of chronic addiction to barbiturates (Danne-man¹⁴) usually take the initial form of elation or euphoria; some cases exhibit depression with suicidal tendencies. Gross memory defects, poor attention, comprehension, and judgment are also manifested. Some cases develop an acute toxic delirium with marked visual and/or auditory hallucinations, which may develop into a chronic psychosis with marked mood swings resembling manic-depressive states.¹⁴ This author also emphasizes that there is often much similarity between the picture of delirium presented by barbital intoxication, and that of delirium tremens.

2. Bromides. Acute cases of bromide intoxication are rare, since vomiting will usually preclude a toxic level of ingestion and retention. Chronic intoxication, however, is quite common, ranking next to barbiturates in order of incidence, particularly because of the ease with which this drug can be obtained without prescription. Some patent medicines contain bromide under the designation of "nerve tonic" and thus are capable of producing a cumulative intoxicating effect through their continued use. However, in contrast to barbiturate intoxication, bromide intoxication is only rarely fatal (Alexander, Moore and Leary⁴), (Moore, Alexander, and Ipsen⁴⁵). In the few cases of deaths that have been ascribed to bromide intoxication, close scrutiny of the record reveals numerous complicating features, such as alcoholism, old age, and others (Moore, Sohler, and Alexander⁴⁸).

Mental symptoms in chronic bromism vary greatly from patient to patient, often taking bizarre forms, and may closely simulate a vast range of neurologic-organic and functional mental diseases, namely, cerebral tumor, encephalitis, alcohol intoxication, manic excitement, or the manic form of general paresis (Diethelm¹⁵). Some complications, such as arteriosclerosis, anemia, kidney and cardiovascular disturbances, diabetes, and epilepsy, may produce a great variety of reactions to the drug (Moore, Sohler, and Alexander⁴⁸). Moreover, since chronic bromism almost always has its roots in some underlying form of mental disturbance, the observer rarely encounters a psychosis produced solely by the action of bromides.

In many cases, delirium will be present with delusions and predominantly visual hallucinations, although auditory, olfactory, and tactile hallucinations may also occur (Diethelm¹⁵). Initial mental symptoms consist largely of drowsiness, impaired memory and attention, disorientation, or, in some individuals, skin rashes which are acneiform in type. The dura-

tion of intoxication averages two to six weeks.⁴⁸ Sudden withdrawal in the course of bromide intoxication will produce marked mood swings from euphoric to depressive reactions, frequently followed by a frank depression which may persist for a few weeks.¹⁵

The development by Wuth⁶⁹ in 1927 of a simple laboratory technique for the quantitative estimation of bromide in the blood has, of course, facilitated the diagnostic procedure. Skin eruption alone is an unreliable criterion, as it occurs in approximately only 25 per cent of the cases (Hanes and Yates,²⁹ Moore, Sohler, and Alexander⁴⁸). As Preu et al.⁵³ have pointed out, deficient diet and dehydration are equally as important factors in producing intoxication as the actual amount of bromide taken.

Therapy. Sodium chloride should be administered promptly upon establishment of the diagnosis of chronic bromide intoxication in order to facilitate the rapid elimination of bromide; adequate fluid intake should be assured during this period to avoid dehydration.

3. Chloral hydrate intoxication in its acute form is characterized by stupor, decline in blood pressure and body temperature, vasodilatation, decrease in the respiratory rate, and cyanosis. Vomiting may also occur. Delirium is occasionally seen in acute states, but is rapidly followed by collapse. Immediate treatment should be initiated by gastric lavage; respiratory and central nervous system stimulants similar to those recommended in barbiturate poisoning should be used; the administration of glucose in solution by the intravenous route will help guard against shock and aid in protecting the liver from irreversible damage.

Slight tolerance, due to chronic use, may occur with chloral hydrate; habituation may closely resemble the clinical picture of alcoholism, but the gastric symptoms may be more severe, and varying forms of skin eruptions may occur (Goodman and Gilman²⁴).

4. Paraldehyde. This drug has a wide margin of safety, and fatalities from its use are rare. Acute intoxication is characterized by the same symptoms as are seen in chloral hydrate poisoning, and similar treatment measures should be employed. The odor of paraldehyde on the breath is a diagnostic aid.

5. Sulfonmethane and sulfonethylmethane (sulfonal and trional) were at one time widely used for their hypnotic effects, but today they are rarely used. The depressant after-effect and minimal analgesic quality of the former makes its use for sedation rather undesirable; moreover, continued use is contraindicated in view of its marked cumulative toxic effects. Acute poisoning may occur as a result of overdosage, and is characterized by uninterrupted sound sleep and prolonged coma, with gradual respiratory and circulatory impairment. Chronic poisoning is caused by continued daily dosage and is characterized by such nervous system symptoms as muscle cramps, depression, polyneuritis, pareses, and ataxia. Gastro-intestinal symptoms may also appear. In severe chronic poisoning, hematoporphyrin will be present, evidenced by the dark port-wine color of the urine. Skin eruptions and fatty degeneration of the liver may also take place. Such symptoms generally persist for several weeks before anuria, collapse, coma, and death ensue. Sulfonethylmethane has the same uses and dangers, but its sedative effect is more rapid (Goodman and Gilman²⁴). Treatment is similar to that recommended for chloral hydrate intoxication.

ALKALOIDS

6. Morphine and Other Opium Alkaloids (Codeine, Dilaudid, Heroin, Paregoric). Acute morphine poisoning may result from attempts at suicide or from clinical overdosage. Infants, the aged, and patients suffering from myxedema with their low basal metabolic rates are particularly susceptible. Addicts or patients suffering from severe pain are especially tolerant of large doses. Whereas other central depressants, such as alcohol, chloral hydrate, and the barbiturates, may simulate the picture of acute morphine poisoning, respirations are characteristically most depressed, the pupils extremely pin-pointed, and the onset of coma most rapid and progressive in morphine poisoning.²⁴ Treatment of acute poisoning is essentially the same as for barbiturate poisoning, with atropine as an additional effective antidote. There is a need for still closer attention to maintaining respiration. Strychnine and picrotoxin are not advisable, since morphine itself stimulates the spinal cord.

Addiction. In chronic poisoning or addiction, the mechanism of tolerance plays the leading role. The depressant effects of morphine and the other opium drugs on the nervous system are abolished with continued use, unless the dosage is progressively increased, but their excitatory effects are not dulled by habituation. Addiction occurs most commonly with heroin, morphine, and cocaine, in that order, and the severity of the withdrawal symptoms follows the same order (Seevers⁵⁹). As Seevers⁶⁰ has demonstrated, the ratio of analgesic potency of these drugs in proportion to the quantity required to produce the same analgesic response is: dilaudid, heroin, morphine, and codeine in that order; while their relative narcotic action is: morphine, dilaudid, heroin, and codeine in that order. Heroin produces the most euphoria and enhanced activity, then morphine, then dilaudid. Codeine produces very little narcosis or euphoria by the subcutaneous route, which was the route employed in these experiments.

Psychotic manifestations, aside from the transient euphoria produced by narcotics, occur not with addiction, but with withdrawal of these drugs, particularly with heroin. It has been suggested by some investigators that psychic factors—chiefly hysteria—may be at the root of such withdrawal symptoms (Light⁴²), but in many instances the symptoms are so severe as to indicate a more profound disturbance in the organic biochemical balance of the nervous system, an “altered nervous correlation” (Tatum and Seevers⁶⁵). Upon withdrawal of the addict’s narcotic supply, irritability, restlessness, weakness, and marked depression may ensue in addition to marked vasomotor disturbances.

Successful management of narcotic addiction requires the combined co-operative efforts of the internist, psychiatrist, and nurse. The substitution of dilaudid, demerol, or methadone for morphine, and its subsequent withdrawal, produces a more prompt withdrawal with less intense and somewhat briefer syndromes of abstinence phenomena. In a series of experiments conducted by King et al.,³⁹ for instance, the weight loss registered after dilaudid withdrawal took place more rapidly than after morphine withdrawal, but the subsequent weight gain was recovered earlier.

The technique for withdrawal which I have used successfully in morphine addiction is the immediate substitution of a corresponding amount of demerol for the daily morphine dose, administered in four divided doses during the first day of hospitalization. During the subsequent

three days each of the four doses of demerol are then gradually reduced by one-fourth of each dose daily, so that on the fourth day of hospitalization one-fourth of the original dose is administered in four divided doses. During the subsequent three days one of the four residual doses is omitted per day, so that on the third day only one of these doses is administered, withdrawal being complete on the eighth day of hospitalization. The period of partial as well as that of complete withdrawal is further eased by giving insulin in subcoma doses (according to the original method of Sakel⁵⁸) if anxiety predominates. If depression predominates during the withdrawal period, and appears to have significance as a personality problem partly responsible for the addiction, I ease the withdrawal period with a series of electroshock treatments; if the patient is also anxious, with combined convulsive-nonconvulsive treatments. Subsequent psychotherapy in the hospital and after discharge is an important part of the endeavor to prevent relapse.

7. Cocaine has been employed as a central nervous system stimulant for centuries by the natives of Peru and Bolivia, who chew the leaves of the tree from which it is obtained (Goodman and Gilman²⁴). Since this drug stimulates the central nervous system from above downward, it is, firstly, a potent stimulant of the cortex, and this effect constitutes its chief desirability to the addict. Like heroin, cocaine produces euphoria and often pleasurable hallucinations, but it may also induce paranoia, and the addict who suffers from delusions of persecution is a potential menace to his community. Formication is commonly experienced by the chronic user; emaciation from loss of appetite, tremors, and convulsions may ensue. A degree of tolerance to cocaine may be developed, but this is less marked than with morphine,²⁴ and habituation in the sense of psychic dependence is more readily demonstrable clinically than true addiction in the sense of physiologic dependence. Nevertheless, abrupt withdrawal will produce the definite unpleasant sequelae of severe depression and persistent delusions. The cocaine user commonly turns to morphine and abandons cocaine, or he may counterbalance the effects of each with doses of the alternate drug, thus tempering euphoria with depression, and depression with euphoria. As Tatum and Seevers⁶⁵ have pointed out, certain addicts will spend much of the day attempting to strike the proper pharmacologic balance between the two.

Because of the wide use of cocaine as a local anesthetic, acute poisoning is frequently manifested. Excitability, restlessness, talkativeness, anxiety, and confusion become evident, with hyperactive reflexes, rapid pulse, and irregular respirations. Delirium, Cheyne-Stokes respirations, and coma precede fatal termination; acute cocaine poisoning typically runs an extremely rapid course, and death may ensue within a few minutes.²⁴

Therapy. The intravenous injection of one of the short-acting barbiturates is indicated in acute poisoning, and respiration must be maintained by artificial respiration when necessary. Goodman and Gilman²⁴ suggest tourniquets to limit the circulation from the site of cocaine absorption whenever possible.

8. Procaine, while much less toxic than cocaine, may effect idiosyncratic responses such as central nervous system stimulation, which can be controlled by the administration of barbiturates. A few cases of rapid cardiovascular collapse followed by death have been recorded.

9. **Cannabis Indica** ("Hashish," "Marihuana," "Bhang") is habitually used the world over for its psychic effects. Its products can be chewed, smoked, or swallowed. While actual addiction does not take place in the sense that abstinence from the drug creates marked withdrawal symptoms, three stages of poisoning from its use have been distinguished (Yawger,⁷⁰ Bromberg¹⁰): (1) A state of acute intoxication, characterized by general euphoria with sensory, motor, and subjective reactions, which may persist from a few hours to a few days. Such intoxication is sometimes marked by transient states of panic, anxiety, hysteria, or depression. Contrary to popular opinion, no evidence is available to corroborate a consistent positive correlation between crimes of violence and marihuana habituation independent of societal and other environmental motivating factors. Glucose by the oral or intravenous route is a fairly effective antidote for acute intoxication. The treatment for addiction is similar to that for alcohol addiction, beginning with complete withdrawal.

(2) Chronic poisoning, leading to a toxic psychosis, characterized by acute agitation or mania, delusions of persecution (Yawger⁷⁰), hallucinations and disordered sensorium, which may persist for weeks or months. Bromberg¹⁰ has reported that, in such cases, the psychotic picture often crystallizes into that of manic-depressive psychosis or schizophrenia; the toxic elements gradually fade away as the underlying functional psychosis ripens. The author stresses the importance of the personality factor in such a development.

(3) Although no corroborating picture has been seen in this country, East European and Asiatic observers have recorded that chronic dementia and deterioration may ensue with prolonged habituation.

10. **Atropine and Other Alkaloids of the Atropine Series.** Poisoning by atropine and related alkaloids such as stramonium, belladonna, or hyoscyamine produces profound states of restless agitation resembling acute endogenous psychotic excitement. The skin is characteristically flushed, warm, and dry, the pulse slow, and pupils dilated. The pulse, however, when large amounts have been ingested, becomes rapid. Speech may be disturbed and vision blurred; swallowing becomes difficult. Ataxia, excitement, delirium, and later coma and death may ensue. An atropine rash may occur, particularly in children. Cases of poisoning may be due to overdose or too prolonged use of drugs of the atropine series, such as are prescribed in the treatment of parkinsonism. Stramonium cigarettes may be another source of overdose. Moreover, children are particularly susceptible to atropine poisoning in the form of seeds and berries containing this drug, such as wild chokecherry and jimson weed.

Drugs of this series may also be used for homicidal purposes, not only in the classical literature (Hamlet's father) but also in modern life. A characteristic case was that of the premeditated poisoning of a woman who was taken by the guilty man to a psychiatric hospital when a marked state of excitation, as a consequence of repeated administration of toxic doses of the drug, had taken place. She expired within twenty-four hours, whereupon postmortem examination and toxicologic study revealed the true cause of her death. The poison murderer attempted to adduce as a mitigating circumstance the fact that he had taken her to a hospital; the judge, however, in summation pointed out that this excuse did not hold, since the murderer had sealed her death warrant in view of the nature of

the hospital to which he had her admitted, because her behavior from that time on was regarded as a consequence of her mental state, and search for and remedy for the poison was hence abandoned before it could be initiated.

This harsh view of the prevailing diagnostic bias of a hospital devoted to one specialty only, made a profound impression on me. It highlighted the responsibility of the psychiatrist in searching out all possible causes in the illnesses of his patients. If doubt exists for establishing a diagnosis in such poisoning, Dameshek and Feinsilver's¹³ method of injecting 10 to 30 mg. of mecholyl hypodermically will aid in demonstrating the presence or absence of atropine intoxication, because the muscarinic effects of mecholyl are abolished in the presence of atropine or its derivatives.

Treatment. Gastric lavage with 4 per cent tannic acid solution is helpful if the poisonous substance has been ingested by mouth, as in the form of choke cherries. Pilocarpine or mecholyl counteracts the peripheral effects and thus may render the patients more comfortable; the dangerous central effects are in part counteracted by morphine, paraldehyde, the short-acting barbiturates, or chloral hydrate. Artificial respiration (pulmotor) and inhalation of oxygen with 5 per cent carbon dioxide should be ready for use when respiratory depression occurs in the advanced stages of the intoxication. Ice bags and alcohol sponges are helpful in counteracting hyperthermia, a symptom particularly prominent in children.

11. Scopolamine (or Hyoscine) Poisoning is similar to intoxication by atropine and its other related alkaloids except for the fact that it has a profound sedative effect. In doses below the sedative threshold, or in states of chronic intoxication, it may create vivid hallucinations. An intern who mistakenly took a dose meant for a patient suffering from parkinsonism subsequently saw funeral processions and heard funeral bells for the rest of the afternoon. Idiosyncrasy is more common with scopolamine than with atropine, and in some individuals ordinary doses may produce marked intoxication (Goodman and Gilman²⁴). The treatment is similar to that of barbiturate poisoning.

12. Strychnine, like cocaine, acts as a central nervous system stimulant, but its primary effect is on the spinal cord rather than the cortex. Accidental acute poisoning constitutes a particular danger for children, who may ingest rat poison or medicinal tablets—particularly cathartics—containing enough strychnine to cause acute symptoms and even fatal results. Strychnine poisoning in New York State between 1926 and 1932 was responsible for almost one-half of the total number of deaths from poisons in children under the age of 5 years (Ross and Brown⁵⁷). Diagnosis of poisoning is usually not difficult. Stiffness of the neck and facial muscles, dizziness, heightened reflex activity, spontaneous muscle twitching leading to spinal convulsions are the usual sequelae. Death may ensue during such a convulsion, sometimes during the first one, caused by apnea, anoxemia, and excessive stimulation followed by paralysis of the medullary centers.

Prompt administration of barbiturates to prevent strychnine convulsions and to protect the medullary centers is the rational therapy in such cases. After convulsions are controlled, gastric lavage (tannic acid 0.5 per cent or potassium permanganate 1:5000) and administration of activated charcoal by mouth is indicated.

13. Mescaline. Experimental acute reversible temporary psychoses can

be produced by the intravenous administration of this drug. Pupillary dilatation, nausea, and vomiting may occur; visual hallucinations, illusions, and perceptual distortions such as alteration of the time sense are noted; euphoria and either occasional depressions or more frequent paranoid reactions may be seen (Hoch et al.³⁵). The southwestern American Indians have used mescal, obtained from cacti, in their religious ceremonies for its psychic effects (Goodman and Gilman²⁴). Hoch et al.³⁶ noted that sodium amylal tended temporarily to reduce or eliminate the effect of mescaline.

14. Caffeine, one of the series of xanthines, acts as a central nervous system stimulant, first affecting the cortex, next the medulla, and lastly the cord. Acute intoxication has been known to occur with 1 or more grams; while 10 gm. has been estimated as a lethal dose (Goodman and Gilman²⁴), there have been no reported fatalities. Acute poisoning is characterized by insomnia, restlessness, and excitement which may progress to a mild delirium. Tinnitus and other sensory disturbances may occur, together with a quickened respiratory and pulse rate. These effects are readily overcome with the administration of central nervous system depressants, in particular, the short-acting barbiturates.²⁴

The question of chronic poisoning from overindulgence in xanthine beverages rests largely on individual idiosyncrasy. Overstimulation of the central nervous system may be reflected in insomnia and restlessness; cardiac irregularities may be manifested, gastrointestinal irritation and diarrhea may occur. Children are more susceptible to caffeine than adults, inasmuch as their normal reflex excitability is high.²⁴ Undoubtedly, habituation to the xanthine beverages does take place, but there is no evidence that this national practice has either harmful or lasting effects (Cheney¹¹).

15. Amphetamine (benzedrine, a synthetic amine) has a potent sympathomimetic effect on the central nervous system. Oral administration produces a temporary elevation of mood, lessened fatigue, increased efficiency, and irritability and sleeplessness. Some individuals react to amphetamine with headaches, palpitation, heartburn, dizziness, and vasomotor disturbances.²⁴ Overdosage invariably results in fatigue and depression after the stimulating effects have worn off. Toxic doses produce extension of the mood elevation; hallucinations, delirium, panic, and suicidal and homicidal urges have been observed. Gastrointestinal symptoms and cardiovascular reactions may also be noted. However, amphetamine appears to have a wide margin of safety; in spite of its continued abuse by the laity as a means of overcoming fatigue and drowsiness, few deaths have been directly attributable to its action (Smith⁶²). The short-acting barbiturates are the most effective antidotes.

16. Ergot (Including Lysergic Acid). The mental and emotional manifestations of ergot poisoning are: confusion, depression, drowsiness, convulsions, hemiplegia, tabetic manifestations, sometimes with a fixed miosis of the pupils. Other symptoms are: headaches, nausea, vomiting, diarrhea, dizziness, weakness, formication, itching, and coldness of the skin. Treatment consists in complete withdrawal of the drug, and the administration of such vasodilators as the cholinesters, including mecholyl, the nitrites, and papaverine.

The experimental production of reversible schizophrenia-like syndromes by lysergic acid has recently been studied by Hoch et al.³⁵ and

Rinkel et al.,⁵⁴ who found that nausea, headache, dizziness, dilatation of the pupils, perspiration and diuresis, ataxia, positive Romberg sign, and forced laughing also ensued with the administration of small oral doses. Sodium amytal counteracted these effects (Hoch et al.³⁶).

17. Nicotine. This drug has very rapid-acting toxic qualities. Deaths have resulted from the accidental ingestion by children of cigarettes and tobacco. Death ensues rapidly from respiratory failure due to the paralyzing effect of nicotine on the respiratory muscles. The treatment of acute nicotine poisoning is primarily aimed at the maintenance of respiration, and artificial respiration or a mechanical respirator should be employed until the crisis is past.

The chronic use of nicotine in the form of tobacco, whether smoked, chewed, or snuffed, is a world-wide indulgence which has been the target of blame for numerous symptoms and complaints. It seems doubtful, however, that the chronic use of tobacco creates a condition of "chronic poisoning" except in individuals with idiosyncratic intolerance. Its use is contraindicated in Buerger's disease and in nicotine amblyopia, and may possibly be contraindicated in other disease syndromes. Habituation is often difficult to overcome. Such psychic abstinence symptoms as continued craving for tobacco, irritability, and restlessness may take place, but are, of course, entirely different from the physiologic dependence seen in drug addiction.

ANALGESIC AND CHEMOTHERAPEUTIC DRUGS

18. Salicylates (Aspirin, Oil of Wintergreen). Mild poisoning by the salicylates is quite common in view of their widespread and often indiscriminate use by the laity. Nephritic patients and children are most susceptible to this drug. "Salicylism"—mild intoxication—when large doses are continually administered will give rise to dizziness, tinnitus, headache, loss of visual acuity, nausea and vomiting, and severe thirst. Mental confusion and symptoms of excitation of the central nervous system, similar to those observed in atropine poisoning, may occur, producing restlessness and incoherence, occasionally progressing to a state of mania with hallucinations or delirium.

Marked similarity exists between the symptoms of diabetic acidosis and salicylate poisoning. Stupor, convulsions, coma, and exitus in respiratory failure may take place in that progression.

Treatment. Arena⁷ recommends gastric lavage, followed by the administration of a saline cathartic. In addition, sodium bicarbonate should be given by mouth in a quantity equal to twice or three times the amount of methyl salicylates that had been ingested. This measure aids greatly in overcoming the toxic effect of the salicylate, particularly in cases of accidental ingestion by children (Stevenson⁶³).

19. Acetanilid and Acetophenetidin are capable of producing intoxication of varying degrees. Like the salicylates, these drugs have been exploited extensively in the patent medicine field, and are widely used by the laity. However, they are considerably more toxic than the salicylates (Goodman and Gilman²⁴), and continued or large doses are even more likely to produce intoxication. Individual variation in susceptibility to these drugs is marked, which adds to risk involved in their indiscriminate administration.

One outstanding symptom presented by acute poisoning in these cases is cyanosis which is due to the formation of methemoglobin and sulfhemoglobin. The skin takes on a bluish color, and diagnosis may sometimes be corroborated on spectroscopic examination of the blood. To be detectable by spectroscopic examination, the amounts of methemoglobin and sulfhemoglobin present must be greater than those sufficient to produce clinically obvious cyanosis. Dyspnea, dizziness, and weakness may ensue. There is a drop in blood pressure; vascular collapse and the shock syndrome may occur; a drug rash may be noted. In severe states, excitement and delirium, followed by depression and stupor, may precede terminal asphyxial convulsions and coma. Fatal cases of acetanilid or acetophenetidin intoxication are exceedingly rare: none have occurred in the last eleven years studied (1938–1948) in Massachusetts (Moore, Alexander, and Ipsen⁴⁵); and only one death was ascribed to an unidentified headache powder during the preceding ten-year period (1928–1937) (Alexander, Moore, and Leary⁴).

Diagnosing a state of chronic poisoning may create a difficult problem in view of the fact that the patient is often found to be using these drugs to relieve the very symptoms induced by their habitual use.²⁴ Pulmonary disease or congenital heart lesions present a similar clinical picture of cyanosis; prominent signs of anemia may also be misleading. A few cases of habituation to acetanilid have been reported (Payne⁵⁰); these have been thought to occur more frequently in neurotic or mentally defective personalities. Mental apathy may be marked in chronic toxic conditions. Payne found through animal experimentation (in dogs) that abrupt withdrawal brought on abstinence phenomena, characterized by states of excitement and panic. While prolonged use of acetanilid may be habit-forming, there is no evidence to substantiate its suggested addictive properties in human beings.

In acute poisoning, lavage and emesis are in order. The patient should be kept warm, and fluids administered intravenously for shock, oxygen therapy for respiratory difficulty, stimulants for circulatory collapse. Methylene blue and alkalis may be administered for methemoglobinemia. A blood transfusion may be indicated in acute severe hemolytic anemia.²⁴

20. Cinchophen poisoning produces the same symptoms as salicylism, as well as certain skin lesions and allergic manifestations; occasionally peptic ulcer may ensue. The hepatotoxic effects of this drug may create symptoms indicating liver involvement similar to those seen in delayed chloroform poisoning.²⁴ Symptomatic and supportive treatment should be administered for toxic cirrhosis due to cinchophen. Glucose and saline solutions should be given intravenously. A low fat, high calcium and carbohydrate diet is in order.

21. Sulfanilamide and Its Derivatives. In its mildest form, sulfonamide intoxication produces dizziness, tinnitus, malaise, headache, and behavioral changes suggestive of mild inebriation. In regard to the latter, there is summation of this effect of sulfonamide with that of alcohol, and for this reason, patients taking sulfonamides should refrain from drinking alcoholic beverages and should not drive a car. Anorexia, nausea, vomiting, and diarrhea may occur. In addition to apparent inebriation, such mental symptoms as depression, confusion, and decreased mental alertness may be seen. Peripheral neuritis may be associated with these mental dis-

appetite. The mood may be elevated to a degree impairing judgment and perspective, at this level grossly resembling alcohol intoxication.⁵⁶

The most effective treatment for the more severe types of ACTH or cortisone-induced psychoses which outlast administration of these substances is electroshock therapy.

Withdrawal of cortisone or ACTH may also elicit psychologic responses; fatigue and exhaustion, asthenia and a general "let-down" may ensue.⁵⁶

NONTHERAPEUTIC ORGANIC CHEMICALS

30. Carbon monoxide poisoning is characterized by anoxia. The progressive symptoms of acute intoxication are: a sensation of tightness across the forehead, throbbing headache at the temples, becoming increasingly severe and accompanied by weakness, loss of visual acuity, nausea and vomiting, collapse, increased pulse and respiratory rate, followed by coma, with intermittent convulsions and Cheyne-Stokes respiration. Death is preceded by depressed respiration, slowed heart rate, and weak pulse.

The typical diagnostic sign is "cherry-red cyanosis"—asphyxia associated with a bright red hue of skin and mucous membranes. Spectroscopic examination reveals this to be due to carboxyhemoglobin. Abundant supply of fresh air is the first emergency measure. Artificial respiration should be administered whenever necessary, but pure oxygen or preferably combined oxygen and carbon dioxide therapy should be instituted at once, or as soon as possible, as well as other supportive measures as indicated. The patient should be kept warm and quiet, the latter to keep tissue requirements for oxygen at a minimum. I regard it as important to continue oxygen therapy not only until the patient regains consciousness, but for considerable time beyond, namely until all traces of carboxyhemoglobin shall have disappeared from the blood. In cases of severe intoxication this may take 36 to 60 hours. After the acute emergency is past, the inhalator may be conveniently replaced by an oxygen tent. Intensive oxygen medication is of paramount importance because it greatly abbreviates the time during which carbon monoxide remains attached to the hemoglobin (Henderson and Haggard,³⁰ Goodman and Gilman²⁴), and thus helps prevent the later development of parkinsonism, which is the most unfortunate after-effect of carbon monoxide poisoning (see Alexander²). Intravenous procaine therapy has proved effective in relieving the stuporous, catatonic, and hebephreniform states during the acute, subacute, and chronic phases following carbon monoxide poisoning. (Justin-Besancon and Laroche,³⁷ Amyes, Ray, and Brockman.⁶) The latter authors recommend intravenous infusions of 500 mg. of procaine hydrochloride, thoroughly mixed with 500 cc. of 5 per cent dextrose in isotonic sodium chloride, to be administered slowly within two hours, after a preliminary skin test for sensitivity.

If, in spite of effective management of the acute states of intoxication, later parkinsonian changes should manifest themselves months or years after the intoxication, these should be treated like cases of parkinsonism of other etiology.

31. Kerosene (naphtha and gasoline) poisoning, due to mistaken ingestion, will produce central nervous system depression, which can be effectively treated in the early states with such stimulants as coramine or caffeine. Gastric lavage should be followed by the administration of an oil demulcent. Blood transfusions should be given if indicated (Arena⁷). In

the event of pulmonary edema, oxygen therapy and the other measures for the treatment of pulmonary edema outlined in Chapter X should be instituted.

32. Tetraethyl lead. The clinical manifestations of tetraethyl lead poisoning resemble alcoholic delirium tremens and encephaloneuropathy, except that horror and fright and their violent psychomotor expressions are more predominant than in the vast majority of cases of alcoholic delirium tremens.

Characteristic physical findings are pallor of the skin, low pulse rate, low blood pressure, and subnormal body temperature. Other physical complications which may ensue are liver damage, myocardial failure, and pulmonary edema. The usual symptoms and peripheral manifestations of plumbism such as occur from poisoning with inorganic lead compounds are largely lacking; lead colic does not occur; stippling in the red cells appears only long after the acute cerebral abnormalities produced by these organic lead compounds. The first prodromal symptoms of intoxication are loss of appetite, restlessness, and unaccustomedly vivid and frightening dreams, which wake the patient repeatedly out of a sound sleep, and sometimes leave him afraid of falling asleep again. Within a few days or a few weeks, depending upon the severity of the intoxication, insomnia, difficulty in concentration, increased talkativeness, tremors, ataxia, marked irritability, and persecutory delusions make their appearance, and finally delirium with hallucinations of a particularly frightening and nightmarish quality, to which the patients react with wild terror. These hallucinations and associated illusions derived from incidental noises and sights on the ward may suddenly send these patients scurrying into a corner with the rapid motion of a frightened small wild animal disappearing into its hole, or leaping impulsively toward or, if unrestrained, through a window, or may keep them standing or lying transfixed in mortal terror shrieking at the top of their lungs in the most bloodcurdling manner for minutes at a time, until laryngospasm or cyanosis supervenes. These agitated patients may fight with the real ferocity of cornered wild animals, to the point of biting those who attend them. This frightened agitation may continue to the end; patients may die shrieking.

The predominantly cerebral manifestations of tetraethyl lead poisoning are due to the marked solubility of lead tetraethyl in fats and lipids, leading to a concentration of the poison in the nervous system and the liver.

The lethal dose of tetraethyl lead varies with the duration of exposure: 5 mg. per liter of respiratory air is fatal after ten minutes; 0.5 mg. per liter of respiratory air if taken for ten minutes daily is fatal after 14 to 20 days. This indicates that tetraethyl lead has a cumulative effect when repeated sublethal doses are administered.

In view of the smallness of the toxic dose, the accumulation of lead in the organs, blood, and urine is less than with inorganic lead compounds.

The largest outbreak of tetraethyl lead poisoning occurred in Ohio and New Jersey in the years 1923-1925 in the early days of manufacture of tetraethyl lead gasoline (Eldridge,¹⁷ Hamilton, Reznikoff, and Burnham²⁸ and Kehoe³⁸). Since then, cases of poisoning have been limited to occasional accidents such as from cleaning and scraping a tank where tetraethyl lead gas had been stored (Bowman and Howard⁸), or from cleaning airplane engines in a closed room with rags that had been soaked in tetra-

ethyl lead gasoline (Stoerring⁶⁴), or pressing clothes that had been dry-cleaned in aviation gasoline (Alexander³). The explanation for the latter source of intoxication is the difference in boiling points of the various components of the tetraethyl lead gasoline mixture. The boiling points for the hydrocarbons which compose aviation gasoline vary from 36° C. for pentane up to 125.8° C. for octane, while the boiling point for tetraethyl lead is 210° C.⁴³ Lead tetraethyl therefore remains in the clothes after the gasoline of the mixture has evaporated, and may be vaporized in relatively high concentration under the touch of the hot pressing iron, to be inhaled by the presser.

Treatment. This includes calcium gluconate to hasten the storage of lead in the bones (2 gm. three times a day), magnesium sulfate in sufficient amounts to keep the bowels open and maintain free diuresis, a high vitamin, high carbohydrate diet with vitamin supplements (multivitamin capsules, three times daily orally, and thiamine chloride, 100 mg. intramuscularly daily) and, last but not least, adequately heavy sedation. Kehoe³⁸ also recommends other saline cathartics as needed, as well as a mixture of sodium bicarbonate or sodium citrate with magnesium oxide and calcium carbonate sufficient to make and maintain neutrality of the urine. In cases with absence of edema, large quantities of water and fruit juices may be allowed. Flury²⁰ recommends the use of parathyroid hormone, vitamins, and liver extract. McCombie⁴³ recommends sodium chloride by mouth as well as vitamins and enzymes which promote the production of gastric juice. Kehoe³⁸ warns against the administration of morphine and chloral hydrate, but on the basis of my clinical experience I feel that sedation with large amounts of paraldehyde (18 cc. or more, as needed) and/or barbiturates (seconal 0.1 gm. and nembutal 0.1 gm. to be repeated as indicated) particularly before bedtime is both admissible and advisable.

33. Carbon Disulfide intoxication produces the following psychic symptoms: Rapid mood swings from a state of exhilaration to irritability and often uncontrollable anger, severe memory defects, insomnia, sleep broken by constant nightmares, and lessening of libido. Carbon disulfide intoxication, both acute and chronic, is a common hazard in the manufacture of viscose rayon. Continuous exposure results in gradual involvement of the peripheral nerves, manifested by such symptoms as the sensation of pins and needles, formication, itching soreness of the fingers, arms, and legs, paroxysmal pain in these extremities, followed by extreme tenderness to pressure of the muscles of these limbs, and the development of areas of hyperpathy with ensuing partial or complete loss of sensation. Fatigue, weakness, loss of visual acuity, and ascending paralysis of the extremities are common, together with marked decrease or loss of reflexes, particularly the corneal and pupillary reflexes. If the basal ganglia are involved, parkinsonism, choreatic hyperkinetic states, and unilateral sensory disturbances may be chronic-progressive sequelae to unchecked, long continued, toxic exposure. Diagnosis may be made difficult by the fact that some cases of poisoning present the characteristic symptoms of brain tumor, multiple sclerosis, or cerebrospinal syphilis. Treatment, apart from removal of the patient from the source of exposure, is purely symptomatic (Penn. Dept. Lab. Ind. 1938⁵¹).

34. Hydrogen Sulfide. Irritation and inflammation of the eyelids, together with eye pain, is a significant symptom of hydrogen sulfide poison-

ing. Edema of the lungs may result if a sufficient quantity is inhaled, and pneumonia is a common aftermath. In chronic poisoning, headache, nausea, and dizziness accompany the initial stimulant, then depressant effect of the fumes on the central nervous system. Artificial respiration, oxygen therapy, and other symptomatic supportive measures (see Chapter X) are emergency treatment measures after acute massive exposure.

35. Carbon Tetrachloride poisoning is capable of producing progressive central nervous system depression in the same manner as chloroform, lastly affecting the medulla. The symptoms of acute poisoning are first a typical inebriation, often followed by convulsions. Depression ensues progressively; severe headache and gastrointestinal disturbances may also occur, and cardiovascular collapse may result from the effect of this drug on the heart action. Carbon tetrachloride is specifically toxic to the liver; calcium and a high carbohydrate diet are valuable preventive measures. Chronic carbon tetrachloride poisoning, usually from industrial sources, may result in cirrhosis and macrocytic hyperchromic anemia. Inhalation of carbon tetrachloride vapors may also cause skin eruptions, conjunctivitis, and bronchitis.

In cases of oral ingestion, immediate lavage should be followed by the administration of saline cathartics; further treatment is symptomatic.²⁴

36. Benzol poisoning, in its acute phase, produces first central nervous system excitation, characterized by restlessness, followed by depression of the central nervous system, death ensuing from respiratory depression. States of chronic poisoning, however, are not productive of direct central nervous system depression, but act principally on the bone marrow. The reaction to the toxic effects of benzol may be delayed for months, thus increasing the difficulty of diagnosis. Aplastic anemia ensues, and granulocytopenia or thrombocytopenia may develop. Dimness of vision may be due to retinal hemorrhages (Hamilton,²⁷ Walsh⁶⁷). Therapy is symptomatic, but the importance of preventive measures in industry is essential.

37. Aniline (Aminobenzine) and Nitrobenzene. Aniline poisoning may cause the following nervous and mental symptoms: vertigo, headache, confusion, weakness, and excessive desire for sleep. There is a preliminary stage of excitement in which the patient may appear inebriated as if by alcohol. Later delirium may ensue, followed by stupor and coma. Nitrobenzene poisoning is similar to aniline poisoning except that the central nervous system depression is more severe. The symptoms of chronic intoxication may include peripheral neuritis,²⁴ central parkinsonian motor disturbances, and obsessive-compulsive phenomena (Adler¹).

The most marked physical sign of aniline or nitrobenzene poisoning is cyanosis due to methemoglobin formation similar to that seen in acetanilid poisoning. Aniline is more toxic to the heart than acetanilid, and cardiac collapse is a more frequent outcome. Cases of poisoning may be industrial, related to the manufacture of aniline dyes, soaps, perfumes, flavoring agents, rubber, or photographic materials, but poisoning may also stem from wearing materials dyed with aniline dyes, or from the use of cosmetics containing aniline or nitrobenzene, since these can be absorbed through the skin. Children in particular may be affected from such sources, such as diapers stamped with aniline dyes, crayons, or shoe polish containing these dyes.

Treatment. The source of exposure should be removed. If vapors have

been inhaled, fresh air and oxygen should be administered; if aniline or nitrobenzene has been swallowed, gastric lavage and emetics should be given. Aniline dyes spilled on the skin should be washed off with ether, alcohol, or water. Supportive symptomatic treatment may be needed in cases of cardiovascular disturbances. Acute anemia may require blood transfusions. If methemoglobinemia is marked, 0.2 cc. per kilogram of body weight of a 1 per cent sterile solution of methylene blue may be injected intravenously slowly over a period of five minutes. This measure, while contraindicated in carbon monoxide poisoning, has been regarded as helpful in methemoglobinemia due to aniline or nitrobenzene poisoning (Arena⁷).

INORGANIC CHEMICALS

38. Arsenic. Acute arsenic poisoning produces acute gastrointestinal distress together with constriction of the throat and difficulty in swallowing, severe thirst, and muscle cramps. Shock may occur, followed by coma and rapid death, usually within twenty four hours or less of ingestion. Gastric lavage with 1 per cent sodium thiosulfate solution, saline cathartics, intramuscular injections of BAL (3 mg. per kilogram of body weight every four hours for 2 days, then twice daily for ten days or until complete recovery), and the continuous administration of fluids are in order; morphine may be administered for pain.

Chronic arsenic poisoning has a gradual onset; lassitude, weakness, anorexia and occasional gastrointestinal flareups are the common initial symptoms. Skin pigmentation and a variety of skin eruptions may take place; stomatitis, excessive salivation, falling out of the hair and nails, edema, and jaundice are symptoms of increasing severity. With the advance of the intoxication, peripheral and central nervous symptoms become manifest. A characteristic state of apathy is seen in severe cases, as well as predominant motor paralyzes of neuro-radiculo-myelopathic type, suggesting Landry's ascending paralysis or the Guillain-Barré syndrome. Determination of arsenic in the urine (and in cases of suspected homicide in the home, of the patient's specially prepared "favorite" tomato juice) are the most important aids in diagnosis, particularly when there is no satisfactory history of arsenic exposure. Removal from the source of poisoning and repeated courses of BAL are the most important items of treatment, as well as such symptomatic means as blood transfusion in the event of aplastic anemia, and respirator ("iron lung") treatment in case of involvement of the upper cervical segments of the spinal cord and its roots.

39. Lead. Chronic lead poisoning results from the slow cumulative absorption of lead salts and is a common industrial hazard, usually incurred by inhalation. Plumbism may be characterized by a few or several of the following symptoms: restlessness, moodiness, excitability, particularly in the early stages; ashen pallor, the appearance of a "lead line"—a black line in the gums close to the border of the teeth—jaundice, and such gastrointestinal symptoms as colic indicate a more severe intoxication. Early signs of nervous system damage are irritability, headache, dizziness, and insomnia. In severe cases, lead palsy with severe peripheral neuritis and muscle paralysis and lead encephalopathy with signs of increased intracranial pressure constitute evidence of the development of central and

peripheral nervous system lesions. Blood changes—secondary anemia and basophilic stippling of the red blood cells—are a good diagnostic guide in chronic lead poisoning²⁴; lead poisoning may at times simulate frontal lobe tumor or multiple sclerosis. The electroencephalogram in one of my patients who presented a clinical picture suggestive of frontal lobe tumor, showed paroxysmal bursts of high voltage slow waves most marked in both precentral and parietal leads, spreading to the right posterior temporal lobe. There were also spike discharges, scattered or in bursts of 1 to 2 per second, most marked in the right temporoparietal lobe.

In treatment, the source of exposure must be determined and eliminated. Calcium gluconate (2 gm. three times a day) should be administered for the purpose of aiding storage of lead in the bones; in addition patients should be on a high calcium and phosphate diet supplemented by ample amounts of vitamins. Additional symptomatic therapy may be administered as indicated. In the presence of encephalopathic symptoms, de-leading procedures, such as by administration of ammonium chloride, 0.5 gm. four times a day, are contraindicated, because these will increase the lead content of the blood, thus exposing the brain to increased amounts of the toxic substance with resulting aggravation or reprecipitation of the cerebrospinal symptoms. BAL is less effective in lead poisoning than in arsenical, mercurial, or cadmium poisoning.

40. Manganese Poisoning, in connection with industrial exposure, may, when its dust or fumes are inhaled over prolonged periods, produce a neurologic picture similar to parkinsonism or the Wilson-pseudosclerosis group of illnesses. In some cases, emotional disturbances have been noted, with involuntary laughing or crying (Goodman and Gilman²⁴). Manganese poisoning may also attack the liver. No specific therapy is known, although it would seem that BAL should be tried.

41. Mercury poisoning occurs in both acute and chronic forms. Oral ingestion, if not promptly followed by vomiting, permits only a poor prognosis; hence, immediate gastric lavage with ample amounts of protein-containing material, such as milk and eggs, or with 10 per cent sodium formaldehyde sulfoxalate which combines with mercury, or with 5 per cent sodium bicarbonate, is indicated in such cases, in order to inactivate the mercury (Arenas⁷). BAL should be administered by intramuscular injection as soon as possible, preferably within the first hour of poisoning, but it will still be effective if given after up to 6 hours. The first dose should be 5 mg. per kilogram of body weight; subsequent doses should be 2.5 mg. per kilogram of body weight, of which the next eleven should be administered at four-hour intervals beginning three hours after the first dose, during the first and second day; on the third day two such doses, on the fourth day one dose may be given. Chronic poisoning from repeated exposure, usually seen in industry, is characterized initially by loss of appetite, stomatitis, colitis, anemia, progressive renal damage, peripheral neuritis, and such central nervous symptoms as depression, insomnia, behavioral changes, and hallucinations.

42. Thallium is an extremely toxic metal, and has strong depilatory qualities. Acute poisoning is characterized by gastrointestinal distress, a gingival line similar to that seen in lead poisoning, and nervous system lesions manifested by paresthesias of the extremities, peripheral polyneuritis, and in chronic cases frequently retrobulbar neuritis. Delirium,

convulsions, and death from respiratory involvement may ensue in severe cases of acute intoxication. Complete loss of hair from systemic application is common. Goodman and Gilman²⁴ recommend that the treatment suggested for arsenic poisoning may be effective in thallium poisoning; the source of exposure, of course, should be removed.

WAR GASES

43. Mustard gas is highly toxic, and its effects are of long duration. Exposure produces severe conjunctivitis, vesication of the skin, lung irritation, inflammation of tonsils, throat, vocal cords, and bronchopulmonary tree. Headache, giddiness, thirst, malaise, dizziness, vomiting, epigastric distress, and diarrhea may accompany the above symptoms. Prophylactic measures to remove the irritant ("decontamination") are most important, followed by symptomatic treatment to relieve distress, especially of respiratory passages and skin. Because of the insidiousness of its effect, its clinging quality and persistency, mustard gas is a stress factor of particularly harassing quality that may affect the morale of troops and precipitate neurotic breakdown among them. The most effective preventive measure is thorough indoctrination and training in, familiarity with, and reliance on decontamination procedures, protective devices (clothing, covers, and masks), and first-aid measures.

44. Lewisite (chlorvinyl dichlorarsine) is a vesicant and lung irritant similar to mustard gas, but with the added systemic toxicity of arsenic. In addition to the protective and therapeutic measures which are similar to those against mustard gas, treatment with anti-Lewisite (BAL, British anti-Lewisite, 2,3 dimercapto-propanol) is necessary (see p. 464).

45. "Nerve Gases" (parathion, DFP (di-isopropyl fluorophosphate), and other organic phosphates such as TEPP (tetraethylpyrophosphate), HETP (hexaethyltetraphosphate); Gammexane (hexa-chlorocyclohexane), MFA (methylfluoracetate), BIS (p-chlorophenyl); TABUN; SARIN). This formidable group of gases is derived from, and in turn has given rise to the development of powerful insecticides and rodenticides. These gases owe their power maximally to excite and then to paralyze the nervous system to their ability to inactivate important enzyme systems, especially cholinesterase as is the case with DFP, the best known and the outstanding working model of most of these substances, but other enzyme systems as well, such as pyruvase in the case of MFA (Himwich³⁴). In view of the fact that these gases inactivate cholinesterase, their toxic effects are intensely cholinergic, similar to those of muscarine, nicotine, and eserine; one important difference is the fact that while eserine inactivates cholinesterase temporarily, the inactivation of cholinesterase by DFP and the other chemicals of this group is for the most part irreversible, recovery depending in large part on the production of new cholinesterase in the body.³⁴

The symptoms of poisoning by the new "nerve gases" and derived insecticides appear in approximately the following order: gastrointestinal symptoms, such as nausea, abdominal cramps, vomiting, diarrhea; sweating, lacrimation, salivation, miosis of the pupils; muscular twitching, increased fatigability, tightness of the chest with prolonged wheezing expiration, suggestive of asthma-like bronchoconstriction; involuntary defecation and urination, weakness of voluntary motion, giddiness; anxiety, depression, mental confusion, convulsions, paralysis, pulmonary edema, and

death. Wood et al.⁶⁸ point out that the survivors of anticholinesterase poisoning may exhibit mental effects for several days following exposure. In patients with mild exposure there may be giddiness, tension, anxiety, jitteriness, insomnia, and excessive dreaming. With more severe exposure there may be withdrawal, depression, restlessness, tremor, emotional lability, and irrational behavior. The electroencephalogram may show intermittent bursts of slow waves (from 4 to 6 per second) of normal to elevated voltage, especially following hyperventilation. Military commanders and medical officers must give serious consideration to the possibility of panic among combat personnel in nerve gas attacks and take all possible steps to prevent or control it.

Treatment. Atropine sulfate counteracts the parasympathetic visceral effects, magnesium sulfate the peripheral muscular phenomena. The central excitatory effects are counteracted by barbiturates, trimethadione (1 gm. in a 20 per cent solution every 15 minutes up to a maximum of 5 gm.), atropine, parpanit, diparcol, and some antihistamines, including dramamine, benadryl, phenergan, and lergigan. The tolerance for atropine is greatly enhanced in subjects poisoned by the nerve gases (anticholinesterase agents). It is therefore important to administer large quantities; repeated intravenous and/or intramuscular doses of 2 mg. each, every 2 to 3 minutes, may be required, until the symptoms are relieved and signs of atropinization, such as dryness of the mouth, appear. Prolonged periods of artificial respiration, preferably with the administration of oxygen under positive pressure are required because recovery cannot be achieved until production of adequate amounts of new cholinesterase has taken place (Himwich,³⁴ Wood et al.,⁶⁸ Arena,⁷ Heymans,³¹ Essig¹⁸ and others). A positive pressure method of artificial respiration is preferable because the profound flaccid paralysis of the respiratory muscles minimizes the elastic recoil of the chest wall (Wood et al.⁶⁸).

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have taken place. Four combined convulsive-nonconvulsive treatments cleared up the psychotic disturbance, but deepened the apparent memory defect. After four subsequent nonconvulsive electric treatments, her memory was not only restored to its former level of function before shock therapy, but had greatly improved over its level of function prior to the apparent onset of her illness. This response indicated that her disturbance of awareness for memory contents was mainly due to anxiety rather than to organic deterioration. After the fourth treatment, this patient, whose Ziehen test indicated severe memory disturbance before treatment, became able to carry out a multiplication, then to repeat two series of six figures, and then after having been thus distracted, to remember the multiplication correctly.

Emotional overlay reactions also play a role in a good many organic neurologic disorders with nonmental presenting symptoms. No one who critically examines the response of organic neurologic patients to various forms of treatment can escape the realization that improvement of function frequently results from psychologic influences. Psychotherapeutic effort, whether unintentional or deliberately planned, has been of decisive importance in many cases which responded favorably to any form of treatment.

This influence is not always recognized; yet it may account for variations in the observed efficacy of the same drug or other physical treatment in certain organic neurologic conditions reported by various observers, whose only difference lies in what Mesmer might have called their personal magnetism. There is no doubt, for instance, that psychotherapy of a supportive and directly encouraging type plays a great role in the results reported from physiotherapy; it may also account in part for the different degrees of improvement achieved with the same physiotherapeutic procedures in different hands. This personal element is particularly important in the treatment of parkinsonism and multiple sclerosis, two conditions whose responsiveness to various stimuli for better or for worse is so marked that at one time, prior to recognition of their organic causation, they were considered forms of neurosis.

For some time I have been interested in utilizing this principle consciously and in exploring the extent to which it renders such neurologic conditions modifiable in the direction of improvement (Alexander¹). The psychotherapeutic procedures which I have employed have included hypnosis² and waking suggestion, as well as supportive, exploratory, interpretive, and abreactive techniques. When necessary I have supplemented these by physical treatment methods, including convulsive and nonconvulsive electric treatment techniques.

There are three important fields of psychotherapeutic endeavor in the treatment of organic neurologic disorders. The first is the treatment of psychogenic overlay reactions; the second is the bringing about of stimulation and activation of performance aimed at helping the patient to compensate for the organic handicap; the third is the prevention of psychosomatic reverberations which might adversely influence the course of the underlying organic illness.

The Treatment of Psychogenic Overlay Reactions. The treatment of the various types of overlay reactions involves particularly treatment of hysteric, depressive, and anxious overlays. The results are particularly

dramatic and decisive when the overlay is of hysteric type, as is frequently the case. Contrary to standard authoritative opinion, organic neurologic disability and hysteria do not tend to be mutually exclusive; rather, many organic neurologic conditions are not infrequently complicated by superimposed hysteric phenomena. It is sometimes the improvement of the hysteric overlay that makes the difference between an invalid and an active useful individual. A case which serves to illustrate this point is that of a 34 year old married woman, mother of two children, who suffered a hysteric paralysis following her discharge from the hospital after she had been informed that she had been suffering from an attack of multiple sclerosis. I treated this patient with an intensive program including hypnosis, strong waking suggestion reinforced by galvanic and faradic stimulation of nerves and muscles, together with exploratory, interpretive, and insight-promoting psychotherapy, as well as with rehabilitative, remedial, and suggestive physiotherapy. In time, she regained her former state of physical and emotional well being, and normal functioning has been sustained. There is no doubt that in this case a minor attack of what may have been multiple sclerosis or polyneuromyelitis, and which was diagnosed as multiple sclerosis by competent observers, had superimposed upon it, or was followed by an essentially hysteric state which was obscured by the presence of organic neurologic findings and was not recognized until the discrepancy between the severity of her disability and the mildness of the neurologic signs made the diagnosis of hysteria inevitable.

Even when neurologic signs are less minimal, for instance in an occasional case in which a positive Babinski sign was present, the actual disability may nevertheless be proved to be hysteric by its prompt response to suggestion. We must remember that after an organic lesion has undergone a rather complete remission—with or without residual reflex anomalies—the pattern of organic disturbance can become hysterically fixed. Good therapeutic results may be obtained in such patients with severe disabling gait disturbance due to hysteric overlay that persisted although the underlying multiple sclerosis was in a stage of good remission. The motivating severe dependent trends must gradually be overcome by intensive psychotherapy, during which time a feeling of control of the situation must be restored to the patient. We must bear in mind in this connection that many persons with chronic illness regress to dependency patterns, but that they will reassume responsibility when they are given the proper kind of supportive treatment in sufficient measure.

A depressive overlay may play a role very similar to hysteric overlay, but it is less well known and appreciated, and more difficult to diagnose, especially when the symptoms complained of fit into the general pattern of the organic disability.

A neurologic disability constitutes severe emotional stress. It may make life seem not worth living. Everything seems an unsurmountable task to the depressive. Everything is an effort. All burdens are heavy. Even lifting the legs from the bed may seem to require enormous effort. In the presence of organic neurologic signs, such a development in a patient's illness tends to be regarded more frequently by the physician as due to progression of the organic illness than as due to a superimposed depression. This diagnosis of progression of the organic disease then confirms the patient's fantasy that he is now completely paralyzed. The event that

he has for months been anticipating with dread or unconscious longing has occurred. The physician thus confirms the patient's fear and unconscious death wishes, and patient and physician may regard this new development as the beginning of the bedridden ordeal whose secondary gains are polarization of affection and attention on the part of family and friends. Thus all too often an unequivocally positive sign of Babinski may become the license for allowing hysteric or depressive behavior to go unchallenged by therapeutic effort. Like hysteric reactions, these depressive reactions can sometimes be treated effectively by psychotherapy alone, aimed at strengthening drives for survival and health; occasionally, however, especially in older persons, these depressions are of sufficient depth to require treatment by electroshock.

In an illustrative case of my experience, a 59 year old woman developed depression following successful ligation of a large right-sided intracranial carotid aneurysm. This depression remained long unrecognized because it manifested itself at first merely in the form of maintenance and elaboration of certain distressing symptoms of her preceding organic illness. It was not until the retention and elaboration of these former symptoms were evaluated against the background of her improved organic status, and in the light of additional specific depressive symptoms which had made their appearance gradually, that the proper diagnosis of depression was arrived at and electroshock decided upon, which subsequently relieved her depression and restored the patient to her normal social and occupational activities.

Overlays of anxiety are easy to recognize. The patient is agitated and frightened. After each tap with the reflex hammer, he asks the doctor whether he is in very bad condition, if there is any hope. This condition is frequently triggered by alarming notions concerning the nature of the disease, notions not infrequently inadvertently transmitted during medical examination when the physician has not fully envisaged the psychologic aspects of the adaptation required of the patient by an organic neurologic illness. Treatment of these anxiety reactions is often difficult and can never be accomplished by encouragement alone. They require the sort of therapy indicated in anxiety states of other origin, especially since they occur mainly in predisposed individuals and result in patterns similar to those elicited, for instance, by combat stress.

Stimulation and Activation of Performance. The second area for psychotherapeutic effort in the management of organic neurologic diseases is that of reinforcing the patient's capacities for adaptation to and compensation for his disability, which may be accomplished chiefly through encouraging, supportive, and suggestive psychotherapy. It is interesting to observe to what extent even those organic disabilities which are entirely uncomplicated by hysteric phenomena can be modified by purely psychotherapeutic procedures such as hypnosis, for instance (Alexander,² Buell and Biehl⁶), sometimes with only temporary improvement, sometimes—by sustained prolonged effort—with improvement over long periods of time. An example is a case of postencephalitic parkinsonism which occurred in a man whose personality and clinical manifestations appeared as completely free of hysteric traits as any patient studied. Nevertheless, it was possible by force of suggestion under hypnosis to create such a state of stimulation and activation that this patient was able to walk with a fast

easy motion, indistinguishable from normal gait by a casual observer and in a motion picture taken at normal speed. A slow motion film, however, revealed that the basic pattern of his gait was actually still the same type of rigid parkinsonian steppage the patient had shown before hypnosis, except that the greater speed, vigor, and long stride gave the illusion of normal gait. It was of interest to observe that together with the improvement of the gait and the motility of the extremities, animation of his formerly masklike facies also reappeared temporarily. The patient was able to keep up this improved performance for two or three hours after each of the eight hypnotic treatments given in a period of sixteen days.

Increasing the cortical excitability by pharmaceutical measures, such as by giving pitressin, had an effect opposite to that of activation by psychotherapeutic suggestion; it enhanced the parkinsonian disturbance in this same patient. However, after the peak of hydration and associated aggravation of his disturbance had passed, strongly worded waking suggestion produced a much more sustained improvement than that following hypnotism, lasting throughout seven subsequent days of hospital observation, during which time he was practically free from parkinsonian disability, and for seventeen additional days following discharge, i.e., for a total of twenty-four days. Then, following a cold, the patient relapsed.

This case demonstrates that, although the organic disturbance itself was unmodifiable by psychotherapeutic effort, such effort still produced a greater readiness on the part of the patient to deploy his reserves, the added speed and vigor greatly aiding his performance even though the basic pattern of the fundamental disturbance remained unaltered.

I have repeated this observation in numerous other parkinsonian patients, one of whom was a man who had been able to write only in slow, tremulous micrographic manner for the past ten years. During and for a short time after hypnosis, in which strong suggestion was given for steady, easy, large free-flowing pen strokes, he was able to write his name and simple sentences with normal speed and in normal size, either without or with only minimal evidence of tremor.

This treatment was repeated three times with essentially identical results. At his request the patient was then given instruction in a method of autosuggestive relaxation. A technique and exercises based on I. H. Schultz's method were used. Four days later the patient's general condition appeared considerably improved. His writing was remarkably improved, although not quite as good as it had been during the third hypnotic treatment. When additional waking suggestion for free, easy strokes was given, however, writing became better and almost attained the improvement obtained during the third hypnotic session five days earlier. This patient left the hospital greatly improved, although little change in the usual medication, which included rabellon and amphetamine, had been made. He maintained a good deal of this improvement for six years, and he had improved again after relapsing one and a half years ago.

I have produced the same surprising response to suggestion in the case of a 15 year old girl suffering from a subacute encephalitic disease with chorea and epileptic seizures, classifiable as a form of Sydenham's chorea. There was no doubt that this girl's illness was essentially an organic one; there were enlargement of cortical sulci, abnormal cerebral electroactivity, and a moderately increased spinal fluid protein.

Nevertheless, psychotherapeutic measures had a remarkable effect in controlling certain aspects of the present symptomatology. Prior to hypnotic treatments, attempts to write her name yielded merely illegible scrawls. After hypnosis, during which strong suggestion for complete motor control and steadiness was given, her writing improved greatly in that it became legible although still awkward and poorly coordinated. Immediately thereafter a psychotherapeutic session was held in which, preliminary to exploration of her anxieties, reassurance was given that all conferences with the physician would be confidential and no details revealed to her parents; immediately after this reassurance her writing was restored to its normal coordination. All three of these writing attempts were made within the same hour. The improvement of her writing remained sustained, and the entire choreiform hyperkinesia abated during the subsequent week. Her seizures were later controlled by dilantin, phenobarbital, and amphetamine medication, and she made an uneventful and sustained clinical recovery. Her abnormal cerebral electroactivity gradually returned to normal during the following year.

The fact that performance and function in obviously organic conditions relatively uncomplicated by psychogenic overlay can be improved by psychotherapeutic procedures is a point of general importance in interpreting the meaning of such improvement. This point transcends in significance the limited subject matter of this chapter. The fact that psychotherapeutic measures bring about improvement of function and performance in certain mental conditions and so-called psychosomatic diseases has all too frequently and too readily been interpreted as indicating that these conditions are of psychogenic causation. It is my opinion that these effects of psychotherapeutic stimulation and activation of performance in states of organically caused dysfunction have established the fact that such a general conclusion is unjustified, since even in conditions of definitely organic causation, such as multiple sclerosis, Sydenham's chorea, and postencephalitic parkinsonism, improvement of function and performance can be brought about by psychotherapeutic techniques.

Psychotherapy is a nonspecific measure which is helpful in any impairment of function that can be aided by facilitation or mobilization of adaptive mechanisms and by adoption of compensatory mechanisms. The way in which psychotherapy helps in these conditions is further proof of the plasticity of the organism, a plasticity which is most pronounced in invertebrate organisms, but preserved to a remarkable degree in vertebrates and even in man. Responsiveness to psychotherapy, however, can never be regarded as evidence of the psychic causation of the disturbance. This fallacy of diagnosis *ex juvantibus* (i.e., diagnosis by what is helpful) is a fallacy which has been effectively disposed of in other fields of medicine, but it remains a source of error not fully appreciated by many representatives of modern psychiatry. Psychotherapy helps everything that can be aided by mobilization of reserve resources, irrespective of the etiology of the underlying condition. It appears to me that the response to psychotherapy shown by certain mental conditions may be of the same order and does not prove the psychologic causation of the disturbance.

Treatment Aimed at Preventing Psychosomatic Reverberations. The third field in which psychotherapy may be helpful in organic neurologic disease, particularly in certain progressive illnesses, is the prevention of

psychosomatic reverberations, especially of anxious and depressive reactions which may influence the course of the disease, irrespective of whether or not the disease itself is regarded as of primarily psychosomatic origin. This is the least established and understood field of psychotherapeutic activity in these illnesses, but nevertheless it is one which appears significant if one follows the course of such illnesses as multiple sclerosis, for instance, from year to year (Langworthy⁸). As one studies the life history of such patients, one cannot escape the realization that responses to stress elicit aggravations, some of them temporary, some of them progressive, just as is the case with arthritis, for instance. Adrenal exhaustion phenomena which play a role in both these illnesses may be the connecting link between the emotional stress and the resulting aggravation of the organic process.

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INTERPRETATION OF RESULTS OF TREATMENT, FLEXIBILITY OF TREATMENT, AND THE NEED FOR ADEQUATE TREATMENT RECORDS

The evaluation of therapeutic procedures and proper interpretation of results of treatment is a crucial task of all medicine, including psychiatry, and an indispensable condition for rational advance. Therapy that does not submit to constant testing and retesting is bound to become frozen in sterile dogmatism and ritualism.

The first premise for the correct evaluation of treatment results is the recognition and acceptance of the fact that any good therapeutic result in any one patient may be entirely due to fortuitous chance. Consistent repetition of results is an essential feature of true science. Sufficiently large numbers to allow for statistical analysis of treatment results are therefore essential. Such analysis is furthermore only possible when a sufficiently large number of untreated control cases, as well as cases treated by methods other than that under scrutiny, are available, and when these control cases are sufficiently well matched with treatment cases to be truly comparable. In order to be comparable, these groups must correspond not only in terms of diagnosis but also in gradation of severity and duration of illness so that it may be correctly surmised that these groups fall into as closely resembling prognostic categories as possible.

The establishment of diagnostic and prognostic categories such as are available for the evaluation of treatment results in many other groups of illnesses is extremely difficult and this has therefore remained one of the greatest stumbling blocks to the development of rational therapy in psychiatry. The reason for this difficulty is becoming increasingly recognized as the multidimensional nature of mental disorder. Diagnosis in terms of only one dimension or factor is inadequate in that it does not differentiate groups sufficiently uniform and distinct to permit extrapolation in terms of prognosis, both if untreated and if subjected to various forms of treatment. The response to various forms of physical treatment, for instance, can be predicted more accurately if a diagnosis in terms of response pattern of the autonomic nervous system is used as the diagnostic criterion that determines the grouping than if categories in terms of diagnosis of the mental manifestations are utilized. Prognosis for favorable response to electroshock treatment, for instance, is 84 per cent for cases diagnosed as

manic-depressive psychosis, depressed—49 per cent for cases diagnosed as schizophrenia (see Chapter XII). If, however, the groups are arranged as to diagnosis in terms of autonomic response pattern, the differentiation between the group responding favorably to electroshock treatment and that responding unfavorably to electroshock treatment becomes much more clear-cut. Favorable prognosis is 90.9 per cent for the group free from epinephrine-precipitable anxiety but reacting with marked and prolonged fall of blood pressure after mecholyl injection, while it is only 11.8 per cent for the group with epinephrine-precipitable anxiety but free from mecholyl-precipitable blood pressure fall (Funkenstein et al.; see Chapter XII, ref. 75, p. 271).

It is obvious, therefore, that adequate grouping of patients for prognostic purposes must be based on at least two dimensions: namely, the psychiatric diagnosis as well as the diagnosis of the reaction pattern of the autonomic nervous system. It appears that the most profound effect of physical treatments is exerted on the physical dimension of the disease, in this case the autonomic reaction pattern, while the concomitant recovery of the mental state seems to be secondary to regained functioning of the centers of the autonomic nervous system. The functioning of the entire physical being, including the centers of the autonomic nervous system, can, of course, be greatly benefited by improvement of the mental state in response to psychotherapeutic influences, unless the psychosomatic disturbance of the autonomic centers has become so severe that it has become truly autonomous, i.e., out of cortical and hence psychologic control. There is, therefore, a close mutual relationship between the psychologic disturbance and the psychosomatic autonomic effects of the disease; so close that any diagnostic category intended as a group for prognostic study, especially for study of response to a particular treatment, must take both these factors into account, unless it is intended merely as a control study of the effect of a treatment on one particular facet of the illness without regard for other aspects of the illness or other diagnostic categories. Such a group must be truly unselected in all other respects.

It was in this manner that I established the effect of nonconvulsive electric treatment on epinephrine-precipitable anxiety by subjecting an otherwise unselected group of psychiatric patients with epinephrine-precipitable anxiety—whose expected rate of good therapeutic response to purely convulsive electroshock treatment was established to be 36.4 per cent by Funkenstein (see Chapter XII, p. 238)—to combined convulsive-nonconvulsive electric treatment. With this treatment, the rate of complete and/or social remission of this group of patients was raised to 91 per cent, a rate of favorable response equal to that of the prognostically most favorable autonomic group. Thus the prognostically unfavorable element of epinephrine-precipitable anxiety was obviously neutralized by the addition of nonconvulsive electric treatment.

There are numerous other dimensions in addition to those discussed above, including intelligence, energy, physical health, motivation, education, social influences, situational factors, and numerous others. All these must be considered when interpreting treatment results and in making up groups for valid comparison. These multidimensional and multifactorial aspects of mental disease and its treatment highlight the importance of all the members of the psychiatric team—the psychiatrist, the internal

medical consultant, the psychiatric social worker, the psychologist, and, last but not least, the psychiatric nurse.

Another aspect in the evaluation and interpretation of treatment results is the timing of the changes that occur during and after treatment, i.e., their time relationship to the treatment as well as to numerous other changes that go on in the patient's mind and body, in his fantasy life, his wishes, desires, fulfillment, chances for fulfillment, and the numerous changes that go on continuously in his social environment.

A young woman, for instance, whom I treated for a severe and prolonged schizophrenic reaction which occurred shortly after her widowed foster mother, to whom she was attached by deep conscious and unconscious bonds, announced her intention to marry a man whom the girl—in part for good reasons—hated and feared, achieved a good social recovery after insulin coma treatment. Three months later, on the day when she learned that her foster mother had broken her engagement, she suddenly achieved a state of apparent complete recovery, which has remained sustained since. In this case we can justly credit the social recovery to the insulin coma treatment, but not the subsequent apparent complete recovery, although the preceding social recovery may have set the stage for her final further step of improvement. In my treatment statistics I am therefore counting only her social recovery as attributable to insulin coma treatment.

Another point important to recognize is the fact that as treatment progresses, the condition of the patient continually changes, in part of course under the influence of the treatment itself. Thus a treatment that may be helpful to the patient today or this week may no longer be helpful tomorrow or next week, because the condition of the patient may have profoundly changed by that time. There has been too much "one-treatment doctoring" going on in psychiatry, patients being subjected to rigidly devised courses of one kind of treatment without frequent periodic revaluation of their changed conditions and recognition of corresponding indications for a change in therapeutic approach derived therefrom. Thus a certain type of treatment, if continued blindly, as it were, without regard for changed indications, may appear to be ineffective or even harmful if carried on beyond the strictly limited time and range of its usefulness; in fact, the more specific the treatment, the more clearly circumscribed is its scope of action.

I should like to give an example in the case of nonconvulsive electric treatment. Figure 143 A shows the pretreatment Funkenstein test in a severe compulsive obsessive state with schizoaffective features in a 28 year old man. His chief complaint was obsessive preoccupation with the wish and the fear of killing his wife; this preoccupation had been fleeting, intermittent, and dismissable for four years; but continuous and overwhelming for three weeks, with resulting inability to work, to relax, or to sleep.

The Funkenstein test showed marked epinephrine-precipitable anxiety with prolongation and an accessory peak of the blood pressure rise on injection of epinephrine (see Fig. 143 A). There was a paradoxical reaction to mecholyl, in that the blood pressure rose instead of falling (Fig. 143 A). In view of this upward shift of the autonomic response pattern, nonconvulsive electric treatment was clearly indicated. Seven such treatments relieved the patient's anxiety as well as completely abolishing his obsessive-compulsive thoughts and preoccupation; but instead depression came to the fore, in that the patient

appeared gloomy, inactive, and discouraged, and reproached himself for ever having had such terrible thoughts as those which brought him to the doctor—although he acknowledged that these thoughts were now gone. Concomitant with this profound change in the mental picture was an equally profound change in his autonomic response pattern (Fig. 143 B). Epinephrine-precip-

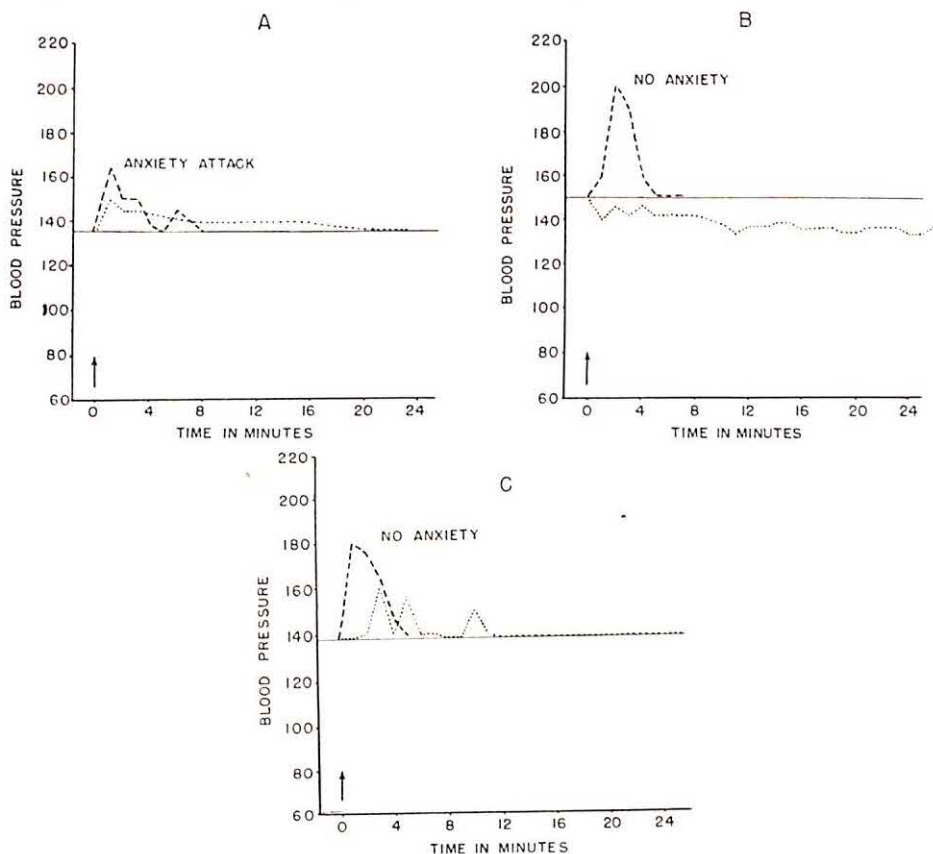


Fig. 143. Male, aged 28, suffering from a severe compulsive-obsessive state with schizoaffective features.

Funkenstein tests. The broken line represents the blood pressure response to epinephrine (0.025 mg. intravenously), the dotted line represents that to mecholyl (10 mg. intramuscularly).

A. Pretreatment Funkenstein test. Note marked epinephrine-precipitable anxiety, as well as a paradoxical reaction to mecholyl, in that the blood pressure rose instead of falling.

B. Funkenstein test after seven nonconvulsive electric treatments. Note absence of epinephrine-precipitable anxiety, and prolonged fall of blood pressure after injection of mecholyl.

C. Funkenstein test after nine subsequent combined convulsive-nonconvulsive treatments, and social recovery. Note continued absence of epinephrine-precipitable anxiety. Injection of mecholyl brought about brief transitory bursts of slight elevation of blood pressure within the first 11 minutes only, a response pattern close to the limits of normal.

itable anxiety as well as the prolongation of reaction to epinephrine had vanished, but injection of mecholyl now brought about a prolonged fall of blood pressure, such as characteristically occurs in typical depressions. I am sure that if nonconvulsive treatments had been continued, his depression would have deepened further. Treatment was therefore changed to combined convulsive-nonconvulsive treatment, the nonconvulsive element being added to prevent reeruption of anxiety and obsessive-compulsive thinking.

After nine such treatments the patient achieved a satisfactory state of social recovery; he was relieved from the rebound into depression by these nine combined convulsive-nonconvulsive treatments, without reexacerbation of the obsessive-compulsive manifestations, from which he had been freed by the preceding series of seven nonconvulsive treatments. After the ninth combined convulsive-nonconvulsive treatment, however, a slight amount of free-floating anxiety recurred in the form of vaguely somatized neurotic concern with himself and expression of worry over the possibility of a relapse at some future date. In spite of this slight rekindling of anxiety, obviously due to the convulsive element of this second phase of the treatment, the patient felt well subjectively, his sleep and appetite were excellent, and he was eager to leave the hospital and to return home and to his work. The Funkenstein test repeated at that time (Fig. 143 C) showed that he was still free from epinephrine-precipitable anxiety; however, there were brief transitory bursts of slight elevation of blood pressure within the first eleven minutes after the injection of mecholyl, a response pattern which may be classified as close to the limits of normal response. Physical treatment was therefore discontinued, and the patient was placed on a regimen of psychotherapy supported by the administration of testosterone (50 mg. three times weekly). The patient achieved a state close to complete recovery one month later, after passing through a phase during which mild epinephrine-precipitable anxiety was rekindled temporarily in the course of psychotherapy, vanishing shortly thereafter in the course of continuing psychotherapy.

This and similar cases emphasize the importance of flexibility of therapy, adapted to the treatment needs of the patient, based on the clinical psychiatric findings as well as on the psychosomatic response pattern, both of which should be frequently reevaluated during the course of treatment. Adequate treatment records are of fundamental importance throughout the course of therapy. A psychiatric reevaluation must be made after every item of physical treatment, and a reevaluation of the psychosomatic-autonomic response pattern whenever the clinical psychiatric picture undergoes a significant change, in order to determine the ever new indication for the next phase of treatment.

A poet once remarked that the gifts of life are thus distributed that, alas, we are given either the wine or the cup, seldom both together. I think that he was referring to the wine of vitality of youth and to the cup of wisdom and experience. I should like to paraphrase this simile, alluding to the increased zest resulting from relief of anxiety and depression, the basic physical change of mood readily afforded by physical treatments, as the wine; and to the understanding of psychodynamics and the appreciation of emotional needs resulting therefrom as the cup. Alas, the wine of physical treatment is too often spilled without the cup. But the cup alone, beautifully designed and crystal clear though it may be, will be void of the substance—the zest of vitality that is often the primary need of the sick person.

In this book, I ventured to outline how wine and cup can be brought together. It is difficult for me to understand why so many adherents of physical treatment should have failed to avail themselves of the cup of psychodynamic understanding, and why so many in the field of psychodynamics should disdainfully reject the wine of physical treatment with a vehemence comparable to that of the crusading ascetic.

Need for Statistical Studies. There is of course a great deal of research to be done in both of these fields, that of physical treatment as well as the psychodynamics of major mental disease. One of the greatest needs is for adequate statistical studies of results. It is surprising how few statistically valid studies are encountered in the literature in reviewing publications of treatment results. The basic need is for uniform discharge statistics (Barton, Tompkins and Nadel⁴); in addition, I think that we psychiatrists should establish standards of follow-up over a period of at least five years, as surgeons do in the case of cancer, or internists in the case of diabetics. Certain aspects of our field compare in some measure to the aspects of care and management of diabetics, since hygienic measures and medical management and treatment become a lifelong problem in either case. Other aspects, for instance those entailed in certain acute psychoses relieved by shock or stimulation treatments or in certain severe chronic psychoses relieved by lobotomy, are similar to the problems of the surgeon.

Some of the inadequacies of our follow-up studies are due to the fact that many of our apparently most effective methods are relatively new developments in our armamentarium. On the other hand, especially in view of the rapid development of new techniques, it is imperative that we carry on adequate statistical follow-up studies so that methods may at

a later date be carefully evaluated. This excuse does not hold entirely for the psychotherapists, since analytic psychotherapy has been in existence for sixty years. I have not encountered a single follow-up study of a significantly large series of patients treated by psychoanalytic methods under optimal conditions, that is, in private practice under the best possible patient-doctor relationship, either for patients with major mental illnesses, or for those suffering from minor emotional aberrations.

I have encountered only a small number of studies based on clinic and hospital material, the patients being treated by a frequent turnover of junior therapists in addition to a smaller number of senior therapists—allowance being made for the fact that treatment conditions were far from optimal—in which follow-up studies extending over one or more years after completion of treatment were carried out (Ross,²³ Miles, Barrabee, and Finesinger,¹⁸ Rennie²⁰ and others). The paper by Miles and his co-workers contains an excellent review of the literature and a convenient table presenting the data available on this important subject. These authors also introduced sound criteria for the evaluation of this problem.

The large series of patients, however, who have been and are being treated under optimal conditions in private practice just do not get into the literature in a form that would make them stand up to critical biometrical appraisal. They are mostly isolated case reports or small groups of cases (Kessel and Hyman,¹³ Hyman¹¹ and others), and those usually with inadequate follow-up. It is of course extremely vexing to have to appraise results in conditions of illness that seem to be at the mercy of an endless variety of external events (Knight¹⁴). On the other hand, some of these events may affect the patient favorably and by and large they should cancel each other out in a sufficiently large number of well observed cases. Besides, it is our aim and in some instances our claim that our treatments can render our patients more stress-resistant, and thus cut down this over-reacting to unfavorable external events. Adequate follow-up studies are our most pressing need for the sake of new and effective developments in our science and art.

Next to it is the need for more harmonious development and integration between the physical, the psychodynamic, and the sociodynamic approaches.

The Neural Mechanisms of Mental Disease. There are, of course, specific developmental needs in each of these specific fields. It has become generally accepted that psychoses most likely belong to the illnesses of stress in the widest sense of the word. There can be little doubt that the stress primarily impinges upon or originates in the psyche or the mental life, and that the initial processes of adaptation, struggle, or exhaustion are going on in the psychodynamic field. There is no doubt, however, that at one point in the chain of events these psychodynamic processes must utilize or express themselves through brain mechanisms, which must be after all the final common path of all processes of thinking, feeling, speaking, and so on. The question has never been solved whether or not these brain mechanisms themselves are abnormal prior to the onset of the psychodynamic struggle which certainly is the precursor of every psychosis, thus putting the individual at a disadvantage by having to fight a complicated campaign with inadequate resources of supply or design of neural power. There is convincing proof that these neural mechanisms become severely

disturbed early in the onset of the psychosis. Funkenstein and his co-workers⁸ discovered the most clear-cut and easily testable set of profound disturbances ascertainable about such neural mechanisms, namely the abnormality in reaction to sympathetic and parasympathetic substances associated with changes in the parasympathetic and sympathetic tone respectively (see Chapter XIII). These authors felt that they could not decide which of the two changes is the primary one, the psychodynamic or the neurovegetative. They stated, however, that they fit together as a hand fits into a glove without stating which is the hand and which is the glove.

Further research in this area is of extreme importance. Since faulty neural mechanisms do exist, their correction would offer great practical therapeutic and preventive opportunities. These neural mechanisms must play a role somewhere, if in no other way than as a final common path. It may be even very helpful, prior to knowing more about the basic mechanisms, to know more about the nature of these final common paths in the brain. While known data are yet greatly insufficient to propound a theory, some physical treatments nevertheless supply a few clues as to what these neural mechanisms concerned with the final common paths in the brain may be. It is of interest to note that electric treatment is ineffective for the relief of depression when it fails to bring about the tonic stretch or phase of a major convulsion which is due to excitation of the subcortical ganglia. The clonic phase which is due to excitation of the cortex adds nothing to the therapeutic effect, and whenever the electrical stimulus has brought about merely cortical excitation such as in nonconvulsive or myoclonic treatment, depression is not relieved at all or may even be aggravated, while anxiety alone is relieved by such a procedure. This observation certainly supplies two important clues, namely that strong stimulation of the subcortical gray matter is needed to relieve depression (which may enhance anxiety), while stimulation of the cortex will relieve anxiety (which may enhance depression). Could it be that in depression either primarily or secondarily an inhibition in the activity of subcortical ganglia has taken place, while cortical activity is relatively enhanced? And could it be that in anxiety cortical activity is inhibited while subcortical activity is relatively enhanced?

The state of the defenses in these two conditions may support these possibilities. There is no doubt that defenses—being a blind unconscious response—are most likely mediated by subcortical structures. These defenses are down in depression, i.e., when we may presume the subcortical structures to be inhibited. They are up in anxiety when we may presume the activity of these subcortical structures to be relatively enhanced. This view may be further supported by the observation that the most marked and profound states of anxiety may be seen in states of severe and acute toxic decortication such as in cases of tetraethyl-lead poisoning (Kehoe,¹² Hamilton, Reznikoff, and Burnham.⁹ Alexander³). On the other hand, severe depression has been observed as an early and sometimes only sign of slow growing thalamic tumors, such as in the case of an astroblastoma of my own experience. Insight is quite obviously a cortical function. Insight is greatly enhanced in certain depressions when defenses are down, while it is always severely inhibited in anxiety states when defenses are up. The incessant ruminations of the severe depressive are additional sug-

gestive indications of a state of cortical excitation that is out of control of the self-protective unconscious forces of the basic subcortical self. When this protective unconscious force, however, runs rampant, as it seems to do in anxiety states, cortical activity may become inhibited. This rudiment of a theory seems to fit the experiences derived from electric treatment. Convulsive electroshock excites the subcortex (presumably inhibited in depression) and may quiet the overactive cortex, thus relieving depression, strengthening the defenses, and lowering the overzealous insight. Nonconvulsive treatment excites the underactive, disorganized, confused cortex and may quiet the overactive, unconscious, subcortical protective mechanisms, thus relieving anxiety, enhancing insight, and lowering defenses. A great deal of research from many angles is needed to see whether or not these few correlated facts and assumptions may lend themselves as foundation stones for further advance in understanding these complex relationships.

Insulin treatment in its earlier phases, and particularly during each immediate post-treatment phase, seems to act like nonconvulsive electric treatment in that it stimulates insight and other cortical activity by lowering defenses, that is, by toning down subcortical activity. In its later phases, and especially in its overall effect, it stimulates basic mood and healthy defense formation in a way suggestive of a stimulating effect upon subcortical centers, without, however, too much inhibition of cortical activity and building up of precipitous defenses, such as occurs with convulsive electric shock. This more general, less unilateral action may account for the greater smoothness but also for the greater slowness of the effect of insulin coma treatment. It appears to restore the balance of both cortical and subcortical activity rather than strengthening one at a time. Insulin subcoma acts more like nonconvulsive treatment in that it relieves anxiety and lowers defenses obviously by enhancing cortical activity and toning down the excitation of the subcortex; however, less intensely because it does not cause a rebound of depression in so doing, which again results in smoother and slower control of the disturbance. The way in which insulin may accomplish these results is of course entirely obscure, except for the fact that profound rearrangement of the nucleoproteins of the nerve cells appears to be taking place (Lorentzen¹⁵). If it could be demonstrated that these changes affect the cortical neurons first, and in subcoma doses foremost, a new means of integrating the resulting psychologic changes with definite levels of the final common path of brain activity may be at hand. This is one of the frontiers on which new data may yield new important territories of knowledge.

From the point of view of this rudimentary theory, lobotomy would merely prevent reverberation between these two relay stations within the final common path. If we view brain circuits as self-reexciting chains of neurons activated by feed-back mechanisms (McCulloch and Pitts¹⁷), we can understand that interruption of the connection may set either relay station at peace. And that is why lobotomy may control either depression with its suggestive subcortical inhibition and cortical excitation, or better still, anxiety and hostility with their surmised subcortical excitation and cortical inhibition.

Autonomic Balance and Mental Disease. The most puzzling territory still is the way in which drugs that stimulate and inhibit the autonomic

nervous system act upon the syndromes of the major psychoses and especially their two basic mood components, namely, anxiety and depression respectively. Funkenstein and his coworkers⁸ have proved that in depression there is an enhanced tone of the sympathetic nervous system associated with increased reaction to parasympathetic (cholinergic) stimuli, while conversely in anxiety the basic tone of the parasympathetic (cholinergic) system is increased, associated with increased reactivity to sympathetic (adrenergic) stimuli. Nevertheless, cholinergic drugs have failed as antidotes to depression and adrenergic drugs have failed as antidotes to anxiety while actually the reverse may be true in slight measure: mechohyl, a cholinergic drug, may sometimes relieve anxiety and amphetamine, an adrenergic drug, relieves depression to a mild degree, although it has no marked or sustained effect especially if the depression has any degree of severity. This seems to indicate that these drugs merely tone down the over-reactivity to the opposing substance, namely that to cholinergic stimulation in depression, and that to adrenergic stimulation in anxiety, thus exerting a splinting or shielding effect as it were, rather than effecting a basic alteration in sympathetic or parasympathetic tone respectively.

The way in which the significant shifts in autonomic balance which characterize successful therapy and recovery of psychotic states take place remains entirely obscure. A marked immediate sweating response is characteristic of most of the therapies employed, particularly of nonconvulsive electric treatment and of insulin treatment, coma and subcoma. It seems to be particularly characteristic of all treatments that relieve anxiety, but it appears also important in electric shock treatment that relieves depression. In my clinical experience I have always taken note of the occurrence, the timing (onset and duration), and the degree of the sweating response. These observations appear to indicate that the antidepressant effect of electric shock treatment was more pronounced whenever the sweating response occurred within the first forty seconds of treatment and was of marked degree. Since sweating is probably a cholinergic response, this would make sense in the light of the fact that the sympathetic tone is increased in depression. This would not explain, however, why this factor is also indicative of a good response to nonconvulsive treatment which relieves anxiety primarily. It also is a marked feature of ether drip infusion treatment (for depression), as well as of such long forgotten treatments as Weir Mitchell's¹⁰ so-called rest cure, during which the patients were wrapped in warm blankets to the point of profuse perspiration, or of such old stand-bys as the pack treatment or the hot bath for states of agitation and excitement, used widely in the past in mental hospitals, prior to the advent of the shock therapies. Veterinarians have also used the age-old principle of the sudorific in treating a state of inert listlessness, akin to a depression, which occurs sometimes in horses. An injection of pilocarpine produces a profound sweating response in the afflicted horse, with oversecretion from the lacrimal and salivary glands as well. Sometimes one such treatment may restore a horse to its former zest.

There is a great need for further studies of the action of drugs in the major psychoses. So far, we understand more about the opposite side of the picture, namely, the experimental production of psychoses by certain drugs, such as mescaline. It is, however, definitely within the realm of possibility that shock treatment may someday be replaced by a variety

of "pills"—one of their ingredients would have to be a powerful cholinergic (parasympathomimetic) substance, probably with marked sudorific side effects.

Frontiers in Psychotherapy. The area of psychotherapy likewise contains vast yet uncharted frontier territories. Over the past sixty years tremendous advances in genetic and dynamic understanding of mental disease have been made. The increased insight into specific problems has led to a more pointed utilization of these insights, making possible the development of more specific therapeutic attacks by brief well planned psychotherapy, such as the brief and intensive analytic psychotherapy developed by Franz Alexander and French,¹ further developed for the specific needs of the psychotic patient by Frieda Fromm-Reichmann,⁷ and with still less orthodoxy of approach, by Rosen.^{21, 22} Thus more specific psychotherapy has become available for specific treatment needs, and the number of known specific dynamic constellations has been considerably expanded since Freud's original discoveries, that fear of loss of love, for instance, plays a decisive role in hysteria, fear of castration and its social derivatives in phobias, and dread of the superego in compulsion neurosis.

Freud's basic insight, namely that symptom formation is substitute formation, has been successfully carried into the understanding of even the major mental illnesses. The basic principle, however, that underlies symptom formation, and more so the source and origin of neurosis, is still the "untouched riddle," as Freud⁶ called it as late as 1936 (p. 92), including even such a well studied and relatively well understood illness as conversion hysteria. Freud said: "What the cause is of the particular obscurity that surrounds symptom formation in conversion hysteria we are unable to guess, but it supplies a reason for quickly leaving this unfruitful field behind" (p. 44). He might have said, instead of unfruitful field, this uncharted territory. Compulsion neurosis, Freud continued (p. 45) "is indeed the most interesting and most grateful subject of analytic investigation, but a problem still unsolved. If we wish to penetrate more deeply into its nature, we shall have to confess that dubious assumptions and unproven conjectures cannot yet be dispensed with." Foremost among these, Freud mentioned the need for assumption of a decisive nonpsychogenic "constitutional factor" to account for the fact that "the genital organization of the libido proves to be too feeble and too little resistant," and that for that reason the "genital organization (of the phallic phase) is wholly or partially thrown back to the earlier anal-sadistic stage." Time factors of onset alone, Freud continues, could not account for this, because "the phallic stage has already been reached when the deviation to compulsion neurosis occurs" (p. 46). In confirmation of this assumption, he mentions a case in which a compulsion neurosis had its onset rather late in life, in obvious response to an actual disturbance of the patient's genitality which had been intact until then. This "supplied the situation prerequisite for regression and for the genesis of the compulsion neurosis." This observation of Freud's is of tremendous theoretical and practical importance. From a practical point of view, it highlights the complexity of the organismal disorganization that we must treat when we are treating the mind. Theoretically, Freud's concept of the genital organization of the phallic phase being too feeble or being disturbed could be expressed more generally in the terms of other schools of psychotherapy as well.

From the psychobiologic point of view, it could be said that the organization of the basic neural systems is too feeble or disturbed; from the Myersonian point of view, one could speak of inadequate or disturbed vitality; and in the terms of Gurdjieff one might speak of insufficient or disturbed harmony of the physical and mental parts of the person.

A most important additional frontier, incursions into which have been made in recent years, outstandingly by the psychodynamic school, by the modern anthropologists, and by social psychiatry, is the field of psychotherapy, especially aimed at prevention of mental illness and deviation by social measures at the levels of family, school, community, law, and government (Levy^{14a}, Zilboorg²⁴). Some progress has been accomplished in what may become a more widespread dissemination of preventive mental hygiene, but we must realize that so far only small beginnings have been made. The next generation of children, brought up in homes and schools influenced by gradually spreading attitudes of better mental hygiene, may enable us to make the first tentative progress report, provided these attitudes are not disturbed by traumatic world upheavals. Even over the past sixty years, certain changes are discernible, although their meaning is yet obscure. Frieda Fromm-Reichmann,⁷ for instance, has pointed out that the most violent defensive attitudes in the present generation of patients are no longer those directed against the sex drives, such as they were in pre-Freudian and Freud's own days, but those directed against hostile and aggressive drives. She feels that the enlightenment brought about by the dissemination of the attitudes of modern psychiatry and mental hygiene has reduced the antisexual taboos to more reasonable proportions, and that the increased recognition of the rights of others inherent in a modern democracy has made the expression of hostility to others, rightly, the worst of the forbidden sins. I fully agree with Frieda Fromm-Reichmann's observation, but I would like to know much more about group dynamics in our society before I would be willing to share her optimistic interpretation. Could it not be that her observation means that there is more widespread regression to anal-sadistic patterns in our competitive and controlled modern society today than there was in the more freely creative (genital-phallic) periods of the past? The emergence of police states with sadistic-aggressive patterns of control, such as Nazi Germany and Communist Russia, certainly points in this direction and should be an effective warning to us.

The unhampered development of the human mind to free, responsible maturity is conceivable only in a free democratic system; and all prevention and treatment of mental illness is based on the recognition of the dignity and the worth of the individual as revealed and preached by all the great enlighteners of mankind.

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